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The Control of Infection in Obstetrics

BY

LEONARD COLEBROOK, M.B., F.R.C.O.G., F.R.S.,
Member of Scientific Staff, Medical Research Council.

Being the William Blair-Bell Memorial Lecture, delivered at the Royal College of Obstetricians and Gynaecologists, January 25th, 1946.

LOOKING back over the recent war years it seems profitable to consider how far the study of wound infections—in particular of streptococcal infections—may contribute to our understanding of puerperal fever.

Before taking up that question I would, however, like to make the point that the coming of penicillin and the sulphonamides does not constitute any ground for complacency in the matter of puerperal infection. The control of streptococcal infection is still a major issue in obstetrics. Although the mortality from these infections is much reduced, their incidence is still too high. From figures kindly supplied to me by Dr. Robert Cruickshank and Dr. Letitia Fairfield, and from the weekly returns published in the *British Medical Journal*, it would appear that there are rather more than 7,000 cases of notified puerperal pyrexia per annum in England and Wales at the present time, and of these, not less than 700 are infections by haemolytic streptococci. It is probable that the true figures would be considerably higher.

It has been suggested that these streptococci are likely to die out under the influence of the new chemotherapeutic drugs. That seems to me improbable for several reasons:

(1) This streptococcus is particularly well adapted for growth in the blood and tissue fluids of human beings.

(2) Although its virulence for man has been declining during the past 40 or 50 years, as shown by the mortality of scarlet fever, there is no assurance that this phase will inevitably continue. We know too little about what determines periodic changes in virulence.

(3) There is now abundant evidence that these streptococci are able to adapt their metabolic processes for survival in the presence of the sulphonamides, and it has been my experience that, when so adjusted, that is, when they have become 'resistant' to these drugs, they are particularly liable to epidemic spread among human beings. That has already happened in several surgical centres

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in this country[1, 2]. In America an important account has recently been published[3] of a major epidemic of streptococcal disease which coincided with the development of sulphonamide resistance in streptococcal strains (Types 19 and 17) in a camp where naval trainees had been given sulphadiazine prophylaxis for the prevention of respiratory infections. It may well be that the next few years will see a wide dispersal of these resistant strains all over the world—an event which might have serious consequences.

No such adaptation to penicillin has yet been recorded, so far as I know, in the case of the haemolytic streptococcus, but since it occurs readily with staphylococcus, both *in vitro* and *in vivo*, we should be rash to assume that it can never happen with streptococci.

Finally, before I leave this aspect of the matter, may I urge the importance of maintaining our vigilance against these unseen enemies of the parturient woman? If we do not go forward in our preventive procedures, we shall probably go backward. We have seen that happen in surgery within the 30 years preceding this last war. In the early days of this century the "no-touch technique" and the careful dressing of wounds with an overall covering of wool as a protective covering to keep out infection were part of the standard teaching of surgeons, but during the first Great War, in the stress of dealing with enormous numbers of casualties, these preventive measures gradually ceased to be standard practice and a generation arose after the war which did not realize their importance. It became necessary to re-introduce a strict no-touch technique in dressings as something new during the last war. In my view it is still our business to improve continually our preventive measures in obstetrics by blocking all the channels along which infection may be transmitted.

To-day I want to speak about one such channel to which we have perhaps paid too little attention, viz. aerial transmission. That possibility has, of course, been more or less in view ever since Lister, following Pasteur, directed attention to its importance in surgery, but the emphasis upon it has fluctuated with the output of evidence—and with the recognition of other possible paths of infection. Thus, between 1890 and 1910 the work of Flügge[4], Koeniger[5], and others on the Continent, and of Mervyn Gordon[6] in this country, convinced many of the inherent dangers of air-borne bacteria; but in 1910 Winslow and Robinson[7], after a long series of experiments, threw some doubt upon its practical importance. At about the same time the risk of contact infection was being re-asserted in surgery; rubber gloves began to be worn; and an aseptic as well as antiseptic ritual in the operating theatre was developed.

In so far as aerial transmission was in men's minds at this time it was chiefly visualized as direct transmission by salivary spray from the mouth. Somewhat later the work of Wells[8] accustomed us to the conception of minute 'droplet nuclei' which could remain suspended in the air and carry infection to considerable distances from the original source. Against these dangers the wearing of efficient masks offered some degree of protection.

Gradually, after the first World War, as bacteriologists made progress in the differentiation of the pathogenic cocci, particularly of the haemolytic streptococci (Dochez[9] *et al.*, Griffith[10], Lancefield[11]), this danger of transmission of organisms from the respiratory tract came more and more into prominence. The wearing of masks became general. In obstetrics, with the work of Smith[12] of Aberdeen, of Hedley Wright and others[13] in London, and in

particular of my sister, Dora Colebrook[14], at Queen Charlotte's, we began to get a body of evidence pointing to the respiratory tract as the probable, or possible, source of many puerperal infections. You will recall that, in 76 per cent of a series of 67 outbreaks or individual cases investigated by my sister, she found a streptococcus serologically identical with that of the mother's genital tract strain in some extra-genital situation—usually the respiratory tract—of somebody, either an attendant, or the mother herself, or a member of her family. My sister refers to these extra-genital situations as *possible* sources of the streptococci which actually infected the mother. I felt at the time that the evidence justified using the word '*probable*', but she was more cautious and preferred '*possible*'. Whatever view we take about the degree of probability, the significance of my sister's findings can hardly be doubted, but she herself would be the first to admit that they did not tell us the whole story. They did not pretend to throw any light upon the question "How precisely did the streptococci pass from the extra-genital place of origin to the genital tract of the mother?" Was it by direct droplet spray from the throat or nose of the individual carrying the organism, or was it by a more indirect route, via somebody's hands or clothes, or possibly by way of dust? We have perhaps been too much inclined to assume that the more direct transmission by droplet spray was the common event, but evidence that has come in recent years suggests to me a different view, namely that indirect transmission has probably played an important part. This is an issue of considerable practical importance, in obstetrics as well as in surgery and medicine, because the planning of appropriate preventive measures largely depends upon it.

If the respiratory tract is the common place of origin of the streptococci, and transfer is usually by direct droplet spray, the wearing of efficient masks should usually be an adequate protection. If, on the other hand, streptococci from the respiratory tract, or from foci of infection elsewhere in the body, are frequently deposited upon dust particles and dispersed through the air in that medium to situations quite remote from their place of origin, then clearly masking will often fail to give protection, and we need to consider measures specifically directed to the elimination of dust in planning for maximum safety in maternity work.

It is this last possibility and the appropriate counter-measures that I want to discuss with you to-day. So far as I know, the first demonstration that haemolytic streptococci may sometimes be found in the air of hospital wards* was given by R. Cruickshank[16]. He showed that the air of the Burns Ward at the Glasgow Royal Infirmary, which housed many patients infected with streptococci, was often contaminated by those organisms, sometimes in considerable numbers; whereas that of the general surgical wards in the same hospital yielded few or none, and he failed to grow any from the air of the medical wards.

At the Isolation Block of Queen Charlotte's we were able to repeat these observations in our single-bedded wards, some of which housed patients with a streptococcal infection while some did not. In a valuable paper "on the possible transmission of haemolytic streptococci by dust"[17], my colleague, Elizabeth White, reported that on all of 27 occasions a series of plates exposed in a ward with a streptococcus-infected patient yielded 1 or more colonies of haemolytic streptococcus (on 22 occasions there were 6 or more), whereas a

*They were reported in the air of military quarters during the pandemic of 1918–19 by Huddleson & Hull[15].

similar series of plates exposed in 6 wards where the patient had not a streptococcal infection yielded only 1 or 2 colonies on 3 occasions. (As the wards all opened upon a common corridor there was ample opportunity for the occasional contamination of a ward by air currents from outside.) Mrs. White's paper also showed that the streptococci found in the air were usually of the same serological type as that of the patient in the ward; and that the number of streptococci on the plates was much increased by sweeping or bed-making; also that they could be recovered at least 5 days after the patient had left the ward (notwithstanding the fact that the rooms were all very light and faced due south). On one occasion, after sweeping out such a ward (5 days after the patient left), Mrs. White herself developed an acute pharyngitis due to a streptococcus of the same type as that isolated from the patient who had been in the room.

Chiefly owing to the arrival of Prontosil (1936) at this time, our attention and energies were diverted for a while and we did not follow up these observations directly upon the patients' bedding. My assistant, Mr. Maxted, did, however, infect the dust of a dark cupboard and showed that in this the streptococci remained alive for at least 10 weeks. He, too, acquired an acute streptococcal tonsillitis and adenitis in the course of this work, and the strain recovered from his throat was identical with the one he had sprayed into the cupboard. The cocci in this cupboard dust retained their full virulence for the mouse for at least 25 days.

Since that time at least 3 papers have reported puerperal fever outbreaks in which the authors thought it probable that dissemination of the streptococci was by way of the air. The first of these, by R. Cruickshank and Godber[18], dealt with 2 such outbreaks. The evidence of aerial transmission was suggestive but not, I think, quite conclusive, since it depended in part upon the assumption that there could have been no faults in the labour ward technique. Haemolytic streptococci were recovered from the air of the wards on one occasion. The outbreak reported in 1944 by Kenny and Barber[19] in the obstetric unit of the British Postgraduate Medical School was of particular interest. In spite of exceptionally thorough and prompt investigation, involving the examination of some 350 swabs, the place of origin of the streptococci was not discovered. These organisms were, however, recovered from the dust in 2 situations, namely the office of the Maternity Unit, and the underside of a lavatory seat which had been used by all but 2 of the 7 mothers who subsequently became infected. This lavatory seat was only 2 feet away from a porcelain slab on which the mothers were bathed before delivery. One can only speculate as to the probable source of the streptococci found in these 2 situations. In view of what we have since learned about the dispersal of streptococci on blankets and clothing, my guess would be that the air of the lavatory and bathroom was polluted by streptococci introduced in this way by one of the antepartum women who was either a nasal or, possibly, a genital tract carrier. (It is of interest in this connection to recall the observations of Thomas[20], who counted the haemolytic streptococci in the dust obtained from the several rooms of a Scarlet Fever Unit and found that the largest number (38 millions) was present in the dust of the bathroom-plus-lavatory annexe.)

This report of Kenny and Barber, like that of Cruickshank and Godber, calls attention to an important fact which we had not previously recognized, that mothers who remain afebrile during the puerperium may sometimes harbour haemolytic streptococci in the genital tract—they are, in

fact, for the time being, genital tract carriers. Unfortunately, none of these cases seems to have been followed up after they left the hospital to ascertain how long the carrier condition persisted.

The third outbreak, which involved some 14 postpartum cases in a private nursing home, was reported in considerable detail recently by Knox and Marmion[21]. Again the ultimate source of the streptococci was not found, but strains of the serological type infecting the mothers were isolated from the air or dust on 8 occasions, and the facts certainly suggest that the dispersal of the organisms by the air may have played an important part in their transmission to new patients.

Apart from these suggestive epidemiological findings, an impressive body of evidence from non-obstetric sources has accumulated during the war, which warrants 2 far-reaching conclusions, viz.:

(1) That pollution of the air by patients discharging streptococci or other pathogens occurs much more commonly, and on a far greater scale, than has usually been recognized.

(2) That bedding and dressings play a large part in this pollution of the air.

I will briefly review some of that evidence. Willits and Hare[22], investigating 14 patients with streptococcus-infected surgical wounds (some of whom were also throat carriers) in Toronto, showed that streptococci of identical type to that of the wound could frequently be recovered from the skin of remote parts of the patient's body, from his clothing, or bed-clothes, as well as from the air surrounding him. Their data support the findings of Wright and Cruickshank[23] as to the dispersal of streptococci by bed-making; and show that a similar dispersal might occur from surgical dressings during their removal. Curiously enough, these workers did not pay much attention to blankets *per se* as a potential source of aerial contamination.

The extensive work of Hamburger and his colleagues[24] who investigated the air of wards housing patients with measles, German measles and other respiratory tract infections, led them to the important conclusion "that the greater number of the haemolytic streptococci in the air probably originate in the 'secondary reservoirs'," i.e. in the floor-dust and bedclothes, and that relatively few are present as 'droplet nuclei' dispersed directly from the respiratory tract. Their paper includes this important sentence: "Cultures of pyjamas, sheets, pillow-cases and blankets of some of these patients have revealed hundreds or thousands of hemolytic streptococci as soon as 6 hours after the patients have been placed in clean bedlinen."

The importance of blankets as potential reservoirs of infection was also studied by Lemon[25], who found no less than 48,000 haemolytic streptococci on one blanket which had been in use on a scarlet-fever patient's bed for 5 days, and 29,000 on a smaller blanket which had been in use for 2 days.

I would like now to show you some visible evidence obtained recently by Dr. Bourdillon and myself in the Burns Unit of the Birmingham Accident Hospital. First I must briefly introduce you to an instrument with which most of you are probably not familiar but which will, I hope, come to form part of the equipment of every large hospital or group of hospitals. This is the 'Slit-Sampler' devised by Bourdillon Lidwell and Thomas[26] for determining the bacterial content of the air. It is essentially a sealed chamber with a narrow slit in its roof through which air is sucked by an electric pump at a definite rate, and made to impinge on the surface of a slowly revolving blood agar plate contained within the chamber (Fig. 1). By this

ingenious instrument we are able to count the number of particles carrying microbes in the air from moment to moment—for example, during the progress of a surgical dressing or an operation.

Such a record of changes taking place in the air during a six minutes' period before and during the dressing of a small burn is shown in Fig. 2. On this occasion bedding was not exposed, so there was no bacterial 'shower' from disturbance of blankets, but the effects of disturbing the bandage and wool are clearly shown. This effect is quite typical of that seen in many similar experiments.

The next series of 'bacteriographs'—as these records might be called—(Fig. 3) shows an extreme example of the bacterial 'scatter' that may occur during the removal of bandages—in this case from a 6-days-old burn of the buttock and thighs of a stout woman. The dressing was slightly moist with exudate (and probably also with urine). Fig. 3 (a) shows the scanty growth of colonies from 40 c. ft. of air before dressings were commenced (1.4 per c. ft.). After the sampling depicted in (a) three small dressings were carried out which raised the bacterial content of the air of the room to between 7 and 14 per c. ft. The result shown in (b) indicates the very heavy contamination of the air which synchronized with removal of the bandages (approximately 154 particles carrying organisms per c. ft.). Most of the colonies are of coliform bacilli and gram-negative cocci (*Diplococcus crassus* type) which were predominant on the patient's burns. The air sample taken half an hour after the dressings were finished (c) and the room left empty shows a complete disappearance of the contaminating organisms and a return to the standard of purity shown in (a).

In Fig. 4 is seen a similar series—before dressing; during removal of bandages from a burn of the shoulders; and again after the room had been left empty for half an hour. In this case the burns were infected with *Staphylococcus aureus* and that organism was present in large numbers on the air plates.

Even higher bacterial densities have recently been encountered while dressings were being done in a Saline-Bath Room; and on almost every occasion the pathogens found on the burned area, some 4-7 feet from the Slit Sampler, were recovered also from the air. Happily we have recently had so few haemolytic streptococci among our burns that I have not been able to get a good record of their scatter in the air, although we have on occasion found a few colonies, just as with other pathogens.

The remaining photographs illustrate the pollution of the air which results from liberation of fluff from blankets and woollen bedclothes. Fig. 5 (a) shows the growth from a 40 c.ft. sample of quiet air and (b) the air of the same room a few minutes later, when the blanket from a patient's bed was momentarily shaken three times at a distance of 3-4 feet from the Slit Sampler. Fig. 6 records the result of shaking (a) a pyjama jacket fresh from the laundry; (b) the same jacket after it had been worn for 3 hours by a patient with burns of the shoulders, completely covered with the usual dressings and a sterilized crepe bandage; (c) the same jacket after it had been worn for 22 hours. A large proportion of the organisms disseminated in the air from this jacket after contact with the patient and his bedclothes were pathogenic staphylococci.

What bearing has all this—and much more evidence of a similar kind—upon the practice of midwifery and of surgery? It brings home to us, surely, that whenever we take down a dressing in an open ward, or in the operating theatre; whenever even pads are changed in a lying-in ward; whenever beds are made; we are in fact

scattering very large numbers of unseen microbes through the surrounding air. The majority of them are harmless types, chiefly micrococci and diphtheroid bacilli, derived from human skin, or are such 'wild' types as are native to the air and soil, e.g. moulds, actinomyces, etc. But not infrequently, in our hospital work, mixed with these harmless types will be a considerable proportion of human pathogens—*Staphylococcus aureus* very commonly, less commonly haemolytic streptococcus, *pyocyaneus*, coliform bacilli, *B. proteus* and perhaps viruses as well. In this connection we do well to remember that about 1 to 2 per cent of all our patients are nasal or heavy throat carriers of haemolytic streptococci; and a much larger number are skin or nose carriers of *Staphylococcus aureus*; so that opportunity must occur very frequently for dispersal of such pathogens from handkerchiefs, clothing, bedding, dressings, etc.

This is a disquieting picture, but we can scarcely shut our eyes to it if we want the maximum of safety in maternity work and in surgery. It becomes still more disquieting when we discover, as I did in Glasgow in 1942, that the standard laundry practice in most of our hospitals does not even attempt the sterilization of blankets and woollen bed-wear. I came upon that knowledge in this way. Finding that something like 80 per cent of the cases in the Burns Unit of the Glasgow Royal Infirmary were becoming infected with haemolytic streptococci within the first few days—many of them on the first day—of their stay in hospital, I began to wonder if some of this infection was being conveyed by the blankets which were put over the patients as soon as they arrived in the ward—often shivering and with no dressing at all over their burns. Cultures from a very small area of 2 blankets (out of 20 examined), fresh from the laundry, yielded haemolytic streptococci of the same type as that which was infecting all the burns. On inquiries being made into the laundry procedure (which I subsequently learned was quite usual in other hospitals) I found that the dirty blankets and other woollens were treated in a 'tumbler' of soap solution at a temperature of 37–43° C. Higher temperatures, such as would have killed haemolytic streptococci and other ordinary pathogens, had to be avoided, I was told, because they caused the woollens to shrink and to become felted. I shall have more to say upon this question later.

The Control of Air-borne Infections.

If I have succeeded in persuading you that contamination of the air by the dispersal of dried dust, visible and invisible, is a common event, and fraught with danger, the question arises: What can we do about it? You will tell me that all our operating theatres, and many labour wards, are purposely designed to avoid just this danger. True, but is that enough? We take care to prevent permanent lodgment of the dust, but have we done all we might to prevent it coming in—on ward blankets, on woollen bed-wear, on theatre stockings, etc.? And have we not, in many theatres, permitted nurses, students and others to come in with outdoor shoes, bringing contaminated dust both from the hospital wards and from the streets? And have we not often ventilated our operating theatres in quite the wrong way—sucking out vitiated air by a fan at roof level and forgetting that this must be replaced by dust-laden air from the corridor, instead of bringing in clean filtered air under positive pressure from the roof of the hospital?

In the wards, both lying-in and surgical, we have scarcely begun to eliminate dust. Dry sweeping is almost universal, but Van den Ende and his colleagues[27] have shown

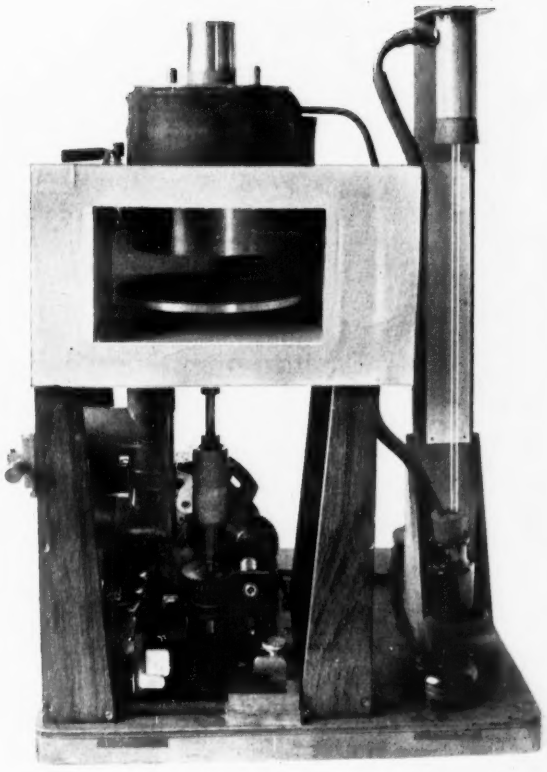

FIG. 1.

The Slit-Sampler (Bourdillon, Lidwell and Thomas)

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DRESSING SMALL BURN OF KNEE

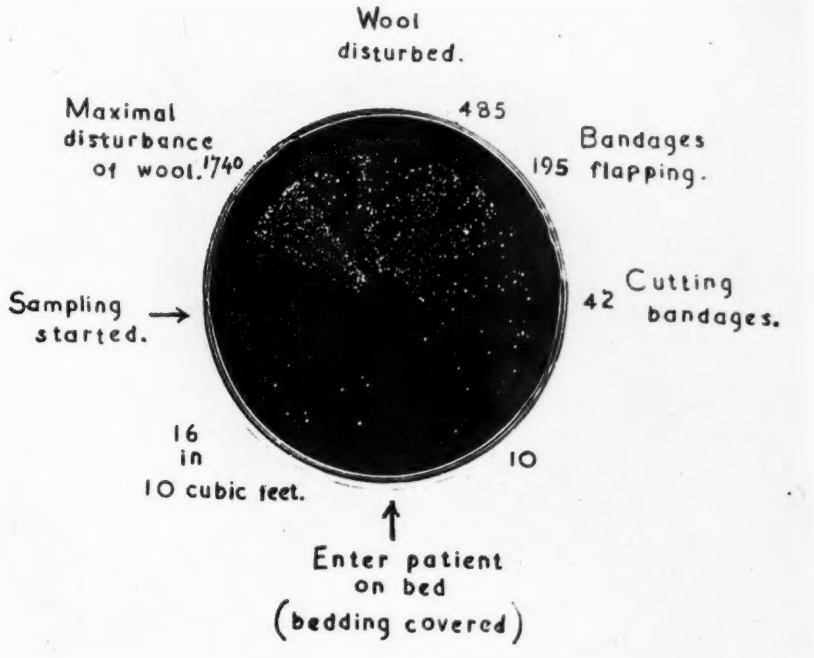
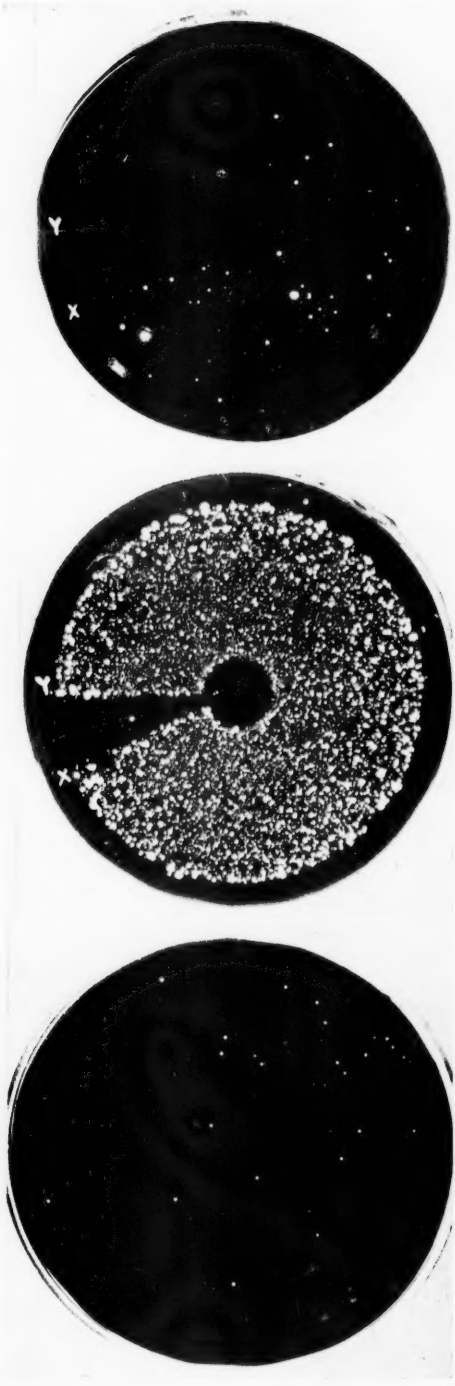


FIG. 2.

Blood agar plate exposed in Slit-Sampler to 40 cu.ft. of air during a 5½-minute run.

FIG. 3.



A. Before dressings were commenced.

B. During removal of bandages from burns of buttocks and thighs.

C. Half-an-hour after dressings finished.

[Sampling started at X and finished at Y.]

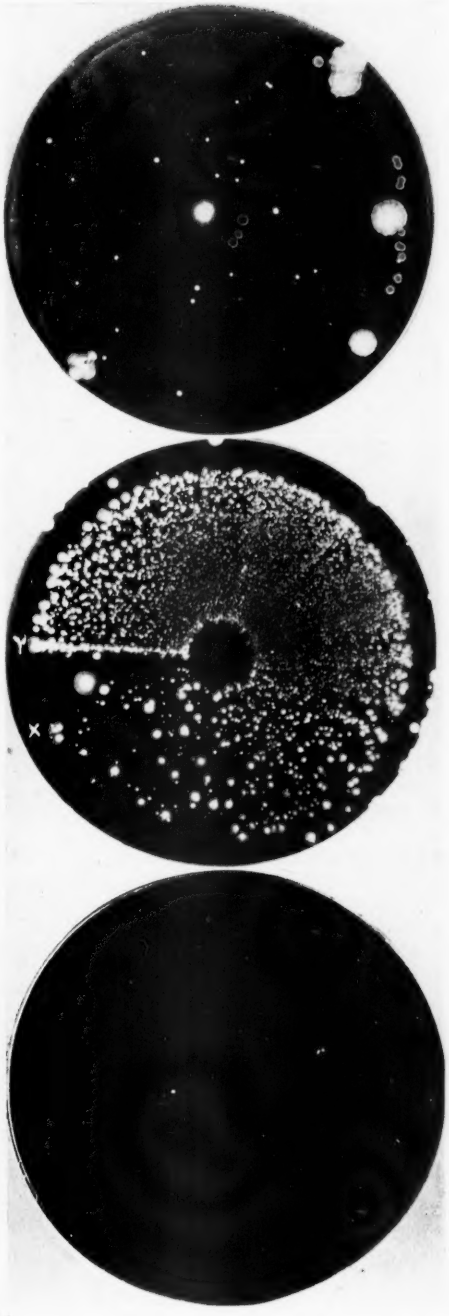


FIG. 4.

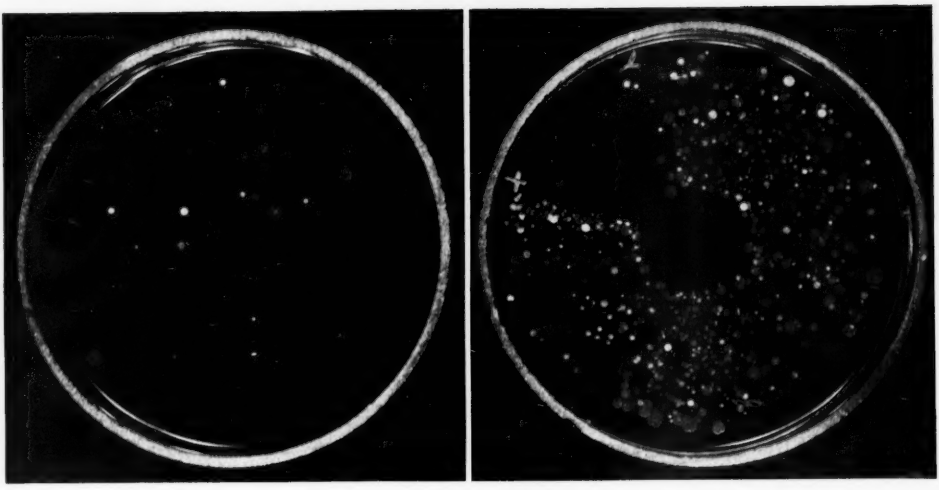
As FIG. 3. Burns of shoulders.

A. Before dressings commenced.

B. During removal of bandages.

C. 15 minutes after patient left the room.

[Sampling started at X and finished at Y.]



(*a*) (*b*)

FIG. 5 (as FIGS. 2, 3 and 4).

(*a*) Air of room before shaking blanket from patient's bed (2 cu.ft.).

(*b*) Air immediately after shaking blanket 3 feet from Slit Sampler (2 cu.ft.).

Sampling started at **X** and finished at Y.

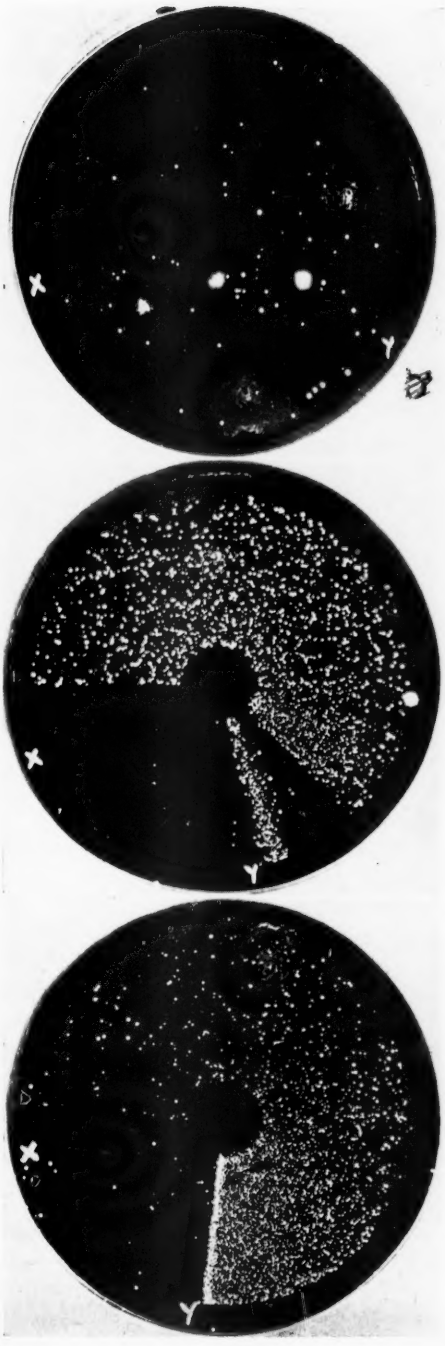


FIG. 6.

Shows organisms liberated from pyjamas.

A. Before being worn.

B. After being worn for 3 hours by patient with burns of shoulders.

C. After being worn by same patient for 22 hours.

[Sampling was started on each plate at X and pyjamas were shaken at Y.]

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that, by the simple expedient of oiling the floors once every 2 to 3 weeks, the dust can be matted so that sweeping will not disperse it through the air; and workers in America have confirmed the great value of this simple measure in reducing pollution of the air. It should, I think, be adopted as standard practice throughout our hospitals.

The same workers have shown, too, that by dipping blankets and woollens in a special oil after they have been laundered, and spinning out the excess in a centrifugal machine so that the oil left is not perceptible to ordinary examination, the liberation of fluff can be very greatly reduced. Owing to shortage of laundry staffs it has been difficult to get this done during the war, but in normal times this, too, might well become a routine procedure. Its great value in reducing cross-infections by streptococci was strikingly shown by Joyce Wright, Cruickshank and Gunn[23], in the measles wards of the London County Council Hospitals.

I spoke earlier about blankets not being sterilized. But they can and should be sterilized. Dr. McCartney and Dr. Cruickshank in the L.C.C. service have shown that they can be steam heated without injury if the pressure is kept down to 5 pounds and the time does not exceed 20 minutes. We confirmed this in Glasgow. Such sterilization will not, of course, kill spores, but it will certainly kill the ordinary non-sporing pathogens such as streptococci.

Blankets can be sterilized in another way, namely by subjection to very strong formalin vapour for 24 hours in a sealed chamber. My colleague, R. M. Fry[28], has shown that it is necessary to use a fairly large quantity of formalin, or, better, paraformaldehyde—50 g. to 1,000 c. ft. of air—and to make sure that the room is perfectly sealed so that the vapour cannot leak away. In my experience this has not always been successful if the blankets are heavily infected with pathogens embedded in an organic matrix such as blood or pus, although for more lightly infected bedding it does suffice.

A third method should be feasible, viz. chemical sterilization during the laundering process, if we could substitute one of the newer antiseptic detergents for the soap usually employed without harm to the blankets. This matter awaits investigation. At present the soap used is chosen for its cleansing properties only.

Finally, I would ask you to think about a more fundamental reform which is worthy of serious consideration in the planning of new maternity and surgical hospitals. I mean the conception that we should do all our obstetrics and surgery in bacteriologically clean air. At present we take great care to prevent anything infected touching the exposed tissues during delivery or an operation, but we are apt to forget that the air touches them all the time and frequently carries pathogens in considerable numbers. I would like to tell you briefly how we have tackled this problem in connection with burns.

My experience in Glasgow had shown me that there was little hope of eliminating cross-infection by streptococci by a rigid dressing technique, masking, etc., so long as the burns were dressed in the open wards, obviously exposed to dust. It seemed clear that a better plan would be to dress them in a special dust-free room, if we could avoid the danger of transmitting the organisms liberated from one patient's dressings to the next. At the Birmingham Accident Hospital we have installed a forced ventilation plant to give us such an abundant stream of warm, filtered air that the whole atmosphere of the room is swept clean about once every 5 minutes. That plan appears to have been successful beyond all our hopes.

In the first year of working since the

plant was installed we have carried out more than 1,400 dressings, usually about 6 per morning, and many of them of extensive burns. Cultures have been taken from each wound every time it was dressed and a complete record has been kept of the order in which the cases were dressed. A very careful scrutiny of these data seems to show that in no single instance has either a haemolytic streptococcus or a pyocyaneus been transmitted in this Dressing Room. A few such added infections have occurred, but the circumstances in every case clearly suggest that the infection occurred in the wards between dressings—no doubt from dust—not in the Dressing Room. That seems to me a remarkable achievement, bearing in mind the past history of burns.

This experiment on ventilation by mechanical displacement of polluted air is of considerable interest. The method is free from the disadvantages attached to sterilization by ultra-violet light and by bactericidal vapours, which have been used in America; and would seem to be more effective than these procedures for eliminating dust-borne bacteria. A ventilation plant such as we have employed in Birmingham need not be a very elaborate or expensive affair provided the air is taken in at a sufficiently high level. Even in the middle of Birmingham the air on the hospital roof is remarkably free from human pathogens.

Whichever technical process is adopted there can be little doubt that continuous ventilation with bacteriologically clean air should be regarded as a reasonable requirement for our labour wards, operating theatres and dressing stations: and also, it may be added, for infant nurseries and premature baby wards. The ventilation of lying-in and other wards is a more difficult problem which I am not competent to discuss.

In conclusion might I briefly suggest to you a few features which should be considered in planning our maternity institutions of the future:

(1) These institutions, if part of a general hospital, should be separate blocks, in order to avoid the possible transmission of infected dust from the surgical or isolation wards by air currents.

(2) There should be a much more generous provision of single-bedded wards than hitherto, in order to allow of the immediate segregation of febrile cases, without waiting for the result of the bacteriological examination. Waiting involves the risk of dissemination of pathogens by bedding, etc.

(3) All wards should be so designed (especially their windows and fireplaces) as to lend themselves readily to sealing with tape for fumigation by formalin or other vapour. This is a point that has not received attention in the past.

(4) Ideally there should be separate quarters for nurses working in the 'clean' wards and in those for febrile cases; but I can see no harm in their mixing when out of uniform. We should remember that the uniform and shoes of medical and nursing staffs must inevitably acquire some contaminated dust from the 'infected' wards. There should be a strict rule about washing of hands before leaving these wards, and throat swabs should be taken before nurses are transferred for duty in 'clean' wards.

(5) Hospital laundries should be equipped for oiling blankets and other woollens and should have separate plants for the washing of bedding from the clean and the infected wards. The soap used in the laundry should be chosen for its bactericidal as well as its cleansing effect.

(6) Provision should be made for the proper ventilation of labour wards, operating theatres and dressing stations with dust-free air.

(7) There should be a 24-hour bacteriological service available in all the larger

maternity institutions, so that swabs can be planted at any hour and a provisional report made without delay. This would mean the provision of a small incubator and the training of at least 2 permanent members of the staff so that they could at least recognize haemolytic streptococci. Their provisional reports must, of course, always be confirmed later by a bacteriologist.

These are the ideas I would leave with you. If you agree that they warrant serious consideration I would add one more that seems to me important. I feel that we need—in some at least of our larger hospitals—a new type of officer whose duty it would be to continually supervise and improve the network of preventive measures we provide. We have been too haphazard and amateurish in the past. Clearly the job is one of applied bacteriology. Such an 'Infection Control Officer' would see to it that the equipment for sterilizing dressings, bedding, bed-pans, etc., is satisfactory, and would carry out periodical tests of its efficiency; he would look into laundry practice; he would organize a service for the supply of sterile syringes with properly sharpened needles; he would advise on the choice of antiseptics and their proper use (a great field for economy). In addition to this and a hundred other matters of daily routine he would advise as to the provision of segregation wards and supervise their fumigation when required; he would be notified automatically of cross-infections occurring anywhere in the hospital and would undertake at once investigations necessary to trace their source; he would ascertain, by periodical tests, that the air supply to theatres, labour wards, etc., is satisfactory, just as, for years past, we have ascertained that water and milk supplies are satisfactory; he would also be responsible for the instruction (with ample practical demonstrations) of students and nurses in all that pertains to the prevention and control of hospital infections.

There can be little doubt that the appointment of a few such officers in our larger hospitals would quickly raise the standard of preventive measures in all the medical services.

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The Value of Antenatal Radiological Pelvimetry

(A Comparative Survey of the Prediction and Event in 300 Successive Pelvimetric Studies at Queen Charlotte's Maternity Hospital.)

BY

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THE value of any method of investigation in practice is clearly dependent upon its reliability and upon the trust that may be placed in the information afforded by it. The study of foetal-pelvic relationships and their influence on the mode of delivery is a study in complex dynamics: by careful antenatal clinical and radiological examination it should be possible to predict with reasonable accuracy the mechanical disadvantages that may arise in the course of any given labour. The influence of 2 important factors, however, cannot be predicted antenatally: these factors are, firstly, the efficiency of the uterine contraction forces, and, secondly, the part played by soft tissues in certain cases.

This paper is a frank attempt to assess the reliability of prediction of the course of labour from antenatal radiological examination alone. It is an attempt to indicate to obstetricians what reliance they may place upon the inferences drawn from a thorough radiological examination by a radiologist with particular interest in, and experience of, obstetric radiology: in the course of the paper it will be shown what has been learnt from a careful follow-up in the borderline cases of disproportion.

In the last 15 years it has been clear from published work that an increasing use has been made of radiological pelvimetry by obstetricians to aid them in their decisions as to the wisest mode of conduct of labour. In earlier years there was much scepticism amongst obstetricians as to the value of X-ray examination. This scepticism is now much less widespread though it still exists. The superb work of Caldwell, Moloy and their colleagues has done much to show how useful radiological examination may be in obstetrics. In recent times in this country it is apparent from published work that certain obstetricians (Chassar Moir,[1] Meave Kenny[2]) place considerable reliance upon X-ray examination, tempered with a dominant clinical judgment. The 2 obstetricians mentioned make their own interpretations from radiographs and their observations are of especial interest because they are, no doubt, directly or indirectly responsible for the conduct of the deliveries. Relatively few obstetricians, however, have made an especial personal study of the scope of X-ray examination, and few have had as intimate an opportunity of assessing its value as Moir and Kenny. We would suggest that the ideal personnel for investigating the problems of disproportion would be a team composed

of an obstetrician interested in the radiological approach and a radiologist interested in obstetrics. Such a team is rarely to be found in practice.

Thus the majority of obstetricians who wish to use radiological methods will have to rely in part on their radiological colleagues for an evaluation of the information that is made available. This paper is intended to show on what grounds clinical judgment should be aided, influenced or amended by radiological findings and the degree of accuracy in prediction that may be achieved.

Clinical Material in the Survey.

The comparison of the radiological prediction and the eventual mode of delivery is based upon a study of 300 pelvimetric cases (examined in the Radiological Department at Queen Charlotte's Hospital between 1942 and 1945) together with a careful follow-up study of each delivery. The series is successive in that it includes every case delivered in Queen Charlotte's Hospital on whom a pelvimetry had been performed. Only those cases delivered in Queen Charlotte's Hospital have been included. Many other pelvimetric examinations have been made in the Department during this time on women who have been delivered elsewhere: e.g. those evacuated from London for reasons arising from the war, those sent up for a consultant's opinion only, and private patients referred to the Department. This group has not been included in this survey because it was felt that only those cases with full recorded notes should be assessed.

Whilst there has not been any radiological selection of the case material, the clinical selection of cases for reference to the X-ray Department has been highly critical. The references have been made for the most part by experienced obstetric consultants (*vide infra*). At no time has radiological pelvimetry been used as a mere routine measure at Queen Charlotte's Hospital. It is fair to state that every case referred has presented a distinct antenatal problem, and in each some guidance or assurance was genuinely sought. This implies that the radiological predictions have been made on a series of patients critically selected on clinical grounds—a necessarily severer test of the value of prediction than if a haphazard group of expectant mothers had been referred for examination. The reader may require to know the status of those referring the cases—they fall into two groups: (1) Consultants (60 per cent of the references) either honorary staff surgeons or antenatal officers; (2) Resident obstetric officers (40 per cent of the references) chosen specifically for their interest and experience in obstetrics and who, almost without exception, have completed the examination for Membership of the Royal College of Obstetricians and Gynaecologists shortly after completing their appointments.

It will readily be agreed that the claim that expert clinical selection of cases for pelvimetry has been made is fully justified.

A routine follow-up study has been made each week (as soon soon as possible after the delivery) from the notes which are kept jointly by the responsible resident medical officer, and the labour ward sister. The standard of record keeping at the hospital is very high. The medical officers are requested to state clearly in their notes the exact indications for any operative intervention. If any point has not been clear from the notes one of us (E.R.W.) has discussed the matter with the officer concerned.

For the purpose of this survey, the correctness of the routinely written radiological predictions (made by E.R.W.) has been assessed by the obstetrician (L.G.P.)

FIG. 1A

W. & P.

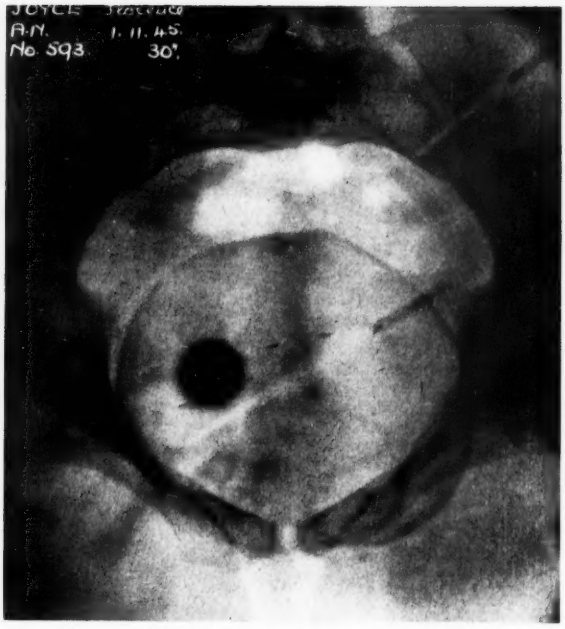


FIG. 1B

W. & P.

FIG. 1C

W. & P.

FIG. 1D

in all instances other than those in which the prediction was "no disproportion, normal delivery" and the event "normal delivery." Any doubtful case has been specifically subjected to the criticism of the obstetrician (L.G.P.).

Technique of Radiological Examination.

The technique described below has been used routinely and fully in every case in the series whatever the reason for the reference of the patient. Not infrequently a pelvimetric examination is requested but not carried out fully when the reason for reference is "high head, difficult to push in." An erect lateral view is taken first; if the head is seen to be engaged in this position, the radiograph is immediately submitted to the obstetrician who then decides whether a full routine examination is necessary. Those cases in which only a lateral view has been taken are not included in this series.

The technique in use at Queen Charlotte's Hospital has been designed to give the maximum information as to the size and shape of the pelvis. It has been elaborated from a method described by Courtney Gage[3] which had been in use at St. Mary's Hospital since 1929. The routine technique has been described fully elsewhere.[4] Briefly, the projections taken are these (Fig. 1 A to D).

1. Anteroposterior of whole pelvis and lower spine, patient supine (stereoscopic pair may be useful).
2. Supero-inferior of the pelvic inlet.
3. Lateral in erect posture.
4. Subpubic arch projection (a modification of Chassard's projection).

In all cases, a true reduced chart of the pelvic inlet, showing its true size and shape, is made (for method, see references 3 and 4). Further, in any borderline case, a similar reduction chart is made from the lateral projection of the medium plane of the pelvis (Fig. 2a.b).

Foetal head models may then be directly compared against these two-plane charts. In specific cases an approximate chart of the bony outlet, as advocated by Moir,[1] may be made though this is an artificial reconstruction and not a single plane reduction from the false to the true size. It is of considerable value, nevertheless, in studying relative proportions in cases with a restricted outlet (Fig. 2d).

The reconstruction-charting just described may be applied to the foetal head after the 34th week of pregnancy. Two projections of the foetal head are obtained and from these, in approximately half the examinations, specific cephalic diameters may be measured. Approximate cephalometry is relatively easy in most cases: accurate cephalometry is difficult in many cases. It is not so much that the size of the shadow of the foetal head is difficult to correct but rather that a precise diameter is often not projected. In any given case, there are 2 technical problems: firstly, is the measurement of the foetal head accurate? Secondly, can one be certain that specific diameters are available for measurement?

Frankly, accurate cephalometry is far from easy to achieve in many cases: is its achievement really necessary? It should be aimed at because obviously for any given pelvis the ideal disproportion-study can be made only with the foetal head that is to go through it. But one has to realize that a foetal head may undergo appreciable diametric changes owing to moulding, and, more important, that there is no way of anticipating with assurance which diameters of the head will present at different pelvic levels; and that the influence of the soft tissues on the rotation, flexion and extension of the head is a factor of great

importance. We are dubious about the value of painstaking cephalometry and and prefer to consider a foetal head as either average, small or large for its maturity. The present attitude is therefore this: when feasible a cephalometry is done and the measurements of what are believed to be specific cephalic diameters are stated: when doubt is felt as to their accuracy, comparison is made against the background of the pelvic reduction charts with models of hypothetical average full-time foetal heads.

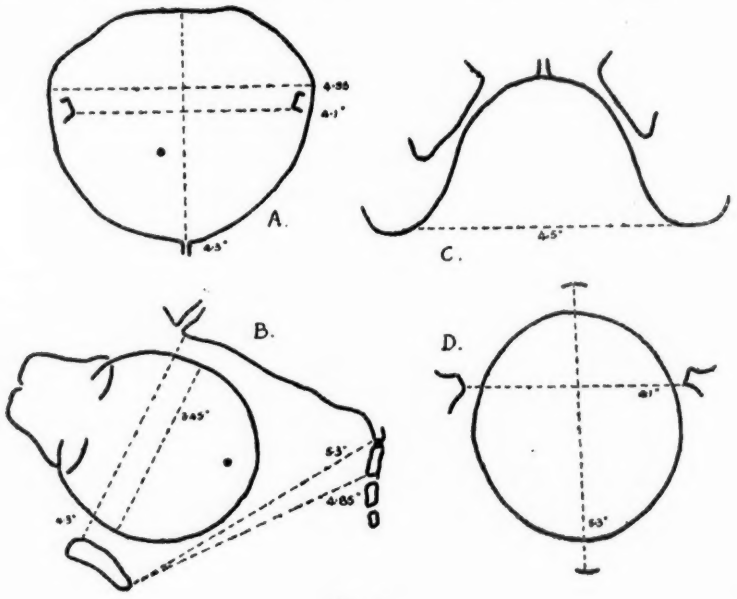


FIG. 2

THE RADIOLOGICAL REPORT AND PREDICTION.

A special report form is used: on it are printed 3 outline drawings of the pelvis to which the diametric measurements are added (Fig. 3). A full descriptive report is given as follows:

(1) The lie of the foetus, the presenting part and the degree of engagement (if any).

(2) In those cases in which a specific cephalic diameter is shown, this is corrected and the true diameter stated: otherwise an attempt is made to classify the head as average, large or small: the degree of ossification is also assessed.

(This applies to cases examined after the 34th week. Predictions made from pelvimetric examinations before the 34th week have assumed an average full-time foetal head. In any case which has been grouped as C, D or E (see later), the head size should be estimated by a second examination as near term as possible.)

(3) A description of the pelvis is given on the lines advocated by Caldwell and his colleagues.

(4) A commentary is written calling attention to significant features, especial consideration being paid to the lower pelvic strait.

(5) The report closes with a prediction, essentially based on relative proportion or disproportion, of the mode of delivery.

cases. Much was also learned from pelvimetric studies made postnatally on patients who had undergone a difficult or aided delivery. Later, predictions were given

QUEEN CHARLOTTE'S MATERNITY HOSPITAL.

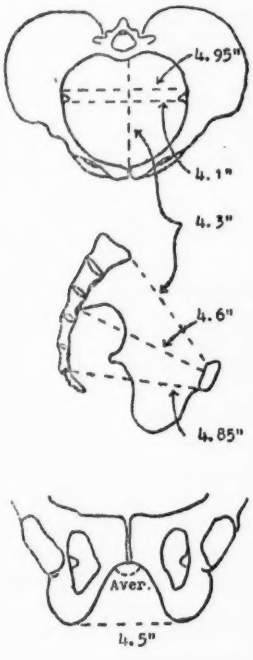
RADIOLOGICAL DEPARTMENT

Name JOYCE, Florence Clinic or Ward Antenatal

Obstetric Officer Miss Robinson Date 1st November 1945

X-Ray No. 593.45

MEASUREMENTS REPORT



FOETUS.

One foetus - Vertex R.O. Lateral.
Head well engaged in erect posture.
Biparietal diameter = 3.45 ins. - average for 36 - 37 weeks maturity.

PELVIC DESCRIPTION.

The inlet is gynaecoid in character.
The side walls show a minor convergence and the ischial spines are not prominent.
The sub-pubic arch is average and is wholly useful.
The sacro-sciatic notches are average-to-wide.
The sacrum is somewhat flat superiorily but slopes well backwards with a vertical coccyx; sacro-coccygeal shelf not prominent.

COMMENTS.

A pelvis of average dimensions showing no unfavourable features in shape. A good obstetric pelvis.

PREDICTION.

No disproportion - normal delivery.

GROUP A.

Radiologist Robin Williams

FIG. 3

It may be asked by virtue of what experience did one of us (E.R.W.) justify giving a prediction routinely. From 1931 to 1941 he gained his experience in obstetric radiology at St. Mary's Hospital, to which patients from Queen Charlotte's Hospital were referred. As a private exercise, from 1934, predictions were made and follow-up studies undertaken in many

openly in the reports—initially to the tolerant amusement of obstetric colleagues —later with their active encouragement. Late in 1941 he was invited to start the radiological service at the new Queen Charlotte's Hospital, and since November 1941 a prediction has been written in every pelvimetric report. It is believed that much more has been learned from errors in writ-

ten predictions (i.e., an official report) than if the prediction had remained a private mental impression.

The Comparative Enquiry.

The Prediction.

The written prognosis or predictions have been groupel under five headings, A to E, as shown in Table I.

Table I.

Prediction Groups.

Group	Relative proportions	Likely mode of delivery	Qualifications
A	No disproportion	Normal	Without reservations
B	No disproportion	Normal	With minor reservations
C	Minor disproportion	Difficult	Normal feasible
D	Moderate disproportion	Abnormal	Normal most improbable
E	Severe disproportion	Abnormal	Normal impossible

Examples of typical reports in each of the 5 groups A to E are given below.

Group A. (No disproportion, normal delivery, no reservations.)

Foetus. Left occipito-lateral; head just engaged in erect posture.

Biparietal diameter = 3.8 inches; average for full-time head.

Pelvic description. A gynaecoid inlet (with minimal android tendencies in the fore pelvis) of ample dimensions. The side walls are straight and the ischial spines are not prominent. The subpubic arch is average ("Norman Arch"). The sacro-sciatic notches are of average width and the sacrum is moderately hollowed.

Comments. A good obstetric pelvis; no disproportion; normal delivery anticipated.

Group B. (No disproportion; normal delivery with minor reservations.)

Foetus. Right occipito postero-lateral; head not engaged in erect posture.

Biparietal diameter = 3.6 inches; average for 37 to 38 weeks.

Pelvic description. A gynaecoid-flat inlet (true conjugate = 3.8 inches). Side walls, straight to divergent; ischial spines not prominent. The subpubic arch is average. The sacro-sciatic notches are rather narrow; the sacrum is well hollowed (capacious hind pelvis in mid-cavity).

Comments. There may be minor difficulty in engagement but once the brim has been entered, descent should be uneventful.

Prediction. Normal delivery with minor reservation re inlet.

Group C. (Minor disproportion; difficult delivery but normal feasible).

Foetus. One foetus, vertex left occipito anterior, not engaging.

Pelvic description. The inlet is almost round, the true conjugate just exceeding the transverse diameter, the latter being well below average (an almost pure transverse contraction). The side walls are straight and the ischial spines not prominent but ischial bispinous diameter = 3.75 inches (less than average). The subpubic arch is average (Norman arch). The sacrum is well hollowed and the sacro-sciatic notches are average-to-wide.

Comments. Probable that engagement and descent will be as direct occipito anterior or occipito posterior. Some arrest on ischial spines likely even with optimal flexion: forceps application will not be easy.

Prediction. Minor outlet disproportion; normal delivery feasible but low arrest anticipated.

Group D. (Moderate disproportion; abnormal delivery, normal most improbable).

Foetus. One foetus, breach left sacro anterior, legs extended.

Pelvic description. Bilateral coxa valga.

An android inlet with a markedly narrow fore-pelvis. Side walls moderately convergent; the subpubic arch is distinctly narrow (Gothic arch), its upper half or two-thirds being useless obstetrically. The anterior-posterior mid-plane and outlet diameters are average. The ischial bispinous and intertuberous diameters are less than average (3.8 inches and 4.05 inches respectively).

Comments. (As for vertex delivery.) To an average full-time head, no difficulty in initial engagement and early descent likely but the narrow fore-pelvis, narrow subpubic arch and the small transverse lower strait diameters make some mid-plane or outlet arrest certain. Not an absolute outlet disproportion but aid by the forceps will surely be required and will be difficult to apply.

Prediction. Moderate outlet disproportion; android pelvis.

GROUP E. (Severe disproportion; abnormal delivery, normal impossible).

Foetus. One foetus, vertex left occipito lateral; head above brim.

Suboccipito-bregmatic diameter = 4.0 inches; and head well ossified, i.e., large baby or post-mature.

Pelvic description. A very small gynaecoid inlet: true conjugate = 3.3 inches; transverse = 4.1 inches. Anterior-posterior diameters in all planes much below average.

Comments. Severe disproportion to even an average head: this head is large and well ossified: engagement improbable and lower capacities very poor.

Prediction. Absolute disproportion at all planes.

THE ASSESSMENT.

(*With explanatory notes for Table II.*)

As previously stated, the assessment of the accuracy of the prediction has been made independently by the obstetrician (L.G.P.). In most instances the assessment has been easily made and the prediction regarded as either correct or incorrect; it is essentially made on the criterion of manifest disproportion (or lack of manifest disproportion) during delivery. It is not based so much on the statement made in Column 3 of Table I ("likely mode of delivery") but rather based on the statement in Column 2 ("relative proportions").

It will readily be appreciated that a Group A prediction of "no disproportion—normal delivery" may be followed by a delivery in which forceps were applied for reasons unconnected with disproportion and the delivery completed without difficulty, e.g. as in the forceps applied for primary uterine inertia or low forceps in a case of cardiac disease. Such a case is not placed in the "correct" column of Table II, but is shown in the column headed "forceps aided—no disproportion" and is finally shown in the "substantially correct" column.

It would clearly be wrong to regard a prediction of "normal delivery" as frankly "correct" when the aid of the forceps was required; but it would also be wrong to regard a prediction of "no disproportion" as frankly "incorrect" in a forceps-aided delivery when no disproportion had been encountered.

A case prediction is regarded as "substantially correct" when delivery was forceps-aided (and easily accomplished) for reasons which are unpredictable from radiological examination.

Predictions which are "Not Assessable."

In Table II is shown a column marked "not assessable". The entries in this column increase in Groups C, D and E, and become maximal in Group E. Almost all of these cases are those in which Caesarean section was performed. A Caesarean section is no test of a prediction of disproportion. For the purpose of this paper, it would have been attractive to have assumed that, because a clinical decision

that a Caesarean section was absolutely necessary, it was therefore justifiable to assume that a radiological prediction of absolute disproportion was correct. This has not been done. Cases delivered by Caesarean section have provided no test of the prediction and are therefore "not assessable." There is no reasonable doubt that in almost all the cases placed in Group E the prediction would have been proved correct.

Percentage Figures.

In Table II the percentage figures are based on the total assessable number, i.e. the absolute total less the "not assessable" numbers.

COMMENTS ON GROUP STATISTICS.

GROUP A.

Total cases, 186. Not assessable, 11. Total assessable, 175.

Correct, 159. Forceps aid, no disproportion, 15. Incorrect, 1.

Substantially correct, 174.

GROUP B.

Total cases, 38. Not assessable, 7. Total assessable, 31.

Correct, 27. Forceps aid, no disproportion, 3. Incorrect, 1.

Substantially correct, 30.

General Observations—Groups A and B.

Of 206 assessable cases in these 2 groups (No disproportion—normal delivery likely)

TABLE II.

Radiological Prediction Groups and Follow-up Assessments.

Prediction groups	A	B	C	D	E	Total all groups
Total cases	186	38	30	25	21	300
Not assessable	11	7	6	8	15	47
Correct	159	27	11	9	4	210
Forceps aided no disproportion ...	15	3	—	—	—	18
Incorrect	1	1	13	8	2	25
Substantially correct	174	30	11	9	4	228
Total assessable	175	31	24	17	6	253
Percentage figures of total assessable	%	%				%
Correct	90.86	87.10				83.0
Forceps aided no disproportion	8.57	9.68				7.11
Incorrect	0.57	3.22				9.89
Substantially correct ...	99.43	96.78				90.11

Percentage figures are not worked out for the individual groups C, D and E, because the total numbers in each of these groups are too small in relation to the whole survey to bear legitimate expression in percentage form.

188 had normal deliveries: 18 cases had forceps-aided deliveries for reasons which could not be predicted and in which there was no evidence of disproportion during the labours. There were 2 faulty predictions, 1 in Group A, 1 in Group B.

Thus of 206 cases, 204 had deliveries without any sign of disproportion. It would therefore appear that, in the Queen Charlotte's Hospital radiological service, whatever the reason for the reference of a patient, if a normal prediction is given it may be accepted with high confidence that normal delivery will occur. The placing of a case in Group A or Group B will justifiably give the obstetrician full assurance that normal delivery is most probable: it will be appreciated that abnormal delivery enforced for reasons other than disproportion cannot be anticipated by radiological but by clinical means.

We feel that the reliability of the prediction of normal delivery alone gives full justification for the use of antenatal radiological investigation. The reader may be reminded that all the patients in this survey were critically selected on clinical grounds.

GROUP C. (Minor disproportion. Difficult delivery).
Total cases, 30. Not assessable, 6. Total accessable, 24.
Correct, 11. Forceps aid, no disproportion, 0. Incorrect, 13.
Substantially correct, 11.

This is a borderline group and, as might be expected, shows the greatest number of incorrect predictions. The fault has clearly been a trend to undue pessimism. In all of the 13 "incorrect" cases, a normal unaided delivery took place without signs of disproportion.

It is of interest that the average birth-weight in the "correct" cases (those in which there was evidence of some disproportion during labour) was 8 pounds, and that the average birth-weight in the "incorrect" cases was 7 pounds 3 ounces.

Has this tendency to undue pessimism been a serious error? We feel that it has not, because at least a sense of awareness for potential dystocia will be fostered in the mind of the responsible clinician. In this Group C there are examples of cases with identical pelvic and cephalic measurements, some showing dystocia, others having easy labours.

In some cases thought to show minor disproportion radiologically, it is clear that with adequate forces and easy moulding, no sign of disproportion has been manifest during labour. With weak expulsive forces, these cases of anticipated "minor disproportion" might have become examples of moderate or even major disproportion clinically.

It is surely the duty of the radiologist to state that minor disproportion is potentially present even though it may not become manifest during labour.

The week-by-week follow-up of cases has certainly helped one of us (E.R.W.) to realize that some features initially regarded as unfavourable need not have been looked upon as necessarily significant. However, it has only been during the work on this survey that the full realization has matured, so that in future work a less pessimistic view will be taken of certain features in the lower pelvic straits. One of us (E.R.W.) has felt strongly that radiologists have an especial responsibility concerning outlet contraction, and in his anxiety not to overlook the significance of minor degrees of outlet contraction undue importance has been given to the sub-average outlet.

The following conclusions concerning the lower pelvic strait have been reached during this survey:

(*a*) The pubosacral or anterior posterior outlet diameter may be as small as 3.75 inches, even with a narrow subpubic arch, without necessarily causing arrest of an average full-time foetal head.

(*b*) The ischial bispinous diameter may be as small as 3.6 inches, even with a narrow subpubic arch, without necessarily causing arrest of an average full-time foetal head.

(*c*) The combination of a narrow subpubic arch, an ischial bispinous diameter of less than 3.8 inches and a pubosacral diameter of less than 4 inches (the lower strait of the funnel pelvis) carries a serious significance.

In Group C, of 25 assessable cases, 22 had either a normal delivery (15) or forceps (7), 2 cases had Caesarean section as an outcome of trial labour, and 1 had "failed forceps and craniotomy." Thus 22 of the 25 cases were not associated with serious danger to the mother. In further practice in this group the majority would be satisfactorily treated as in-patients in a hospital under the care of an obstetric consultant, and a minority might be recommended for trial labour.

GROUP D. (Moderate disproportion; abnormal delivery; normal most improbable).
Total cases, 25. Not assessable, 8. Total assessable, 17.
Correct, 9. Forceps aid, no disproportion, 0. Incorrect, 8.
Substantially correct, 9.

Of the 8 faulty predictions, the bad prognosis in 7 cases was based on outlet considerations and the remarks made above in Group C also apply here. The one faulty prediction concerned with the inlet was given in a case with a true conjugate of 3.45 inches, a child of 6 pounds 2 ounces being delivered without difficulty.

Of the "correct" cases, 2 had Caesarean section enforced after unsuccessful trial of labour, 6 had forceps for midpelvic or low arrest and 1 had a difficult but unaided delivery after induction at the 35th week.

Of the 8 "incorrect" cases, all had normal unaided deliveries, 2 showing unusually strong pains and the average birth-weight was 6 pounds 11 ounces, as against 7 pounds 10 ounces in the "correct" series.

In Group D of the 17 assessable cases, 15 had forceps or normal deliveries unassociated with serious danger to the mother. In future practice, cases placed in this group will be those in which trial of labour would usually be recommended.

GROUP E. (Severe disproportion; abnormal delivery; normal impossible).
Total cases, 21. Not assessable, 15. Total assessable, 6.
Correct, 4. Forceps aid, no disproportion, 0. Substantially correct, 4.

In the majority of cases here with severe or absolute disproportion, elective Caesarean section has been performed. Whilst in the majority the grave disproportion has been recognized clinically, there is evidence that the degree of the severity has not been appreciated in some until the radiological study has been made. In some cases, the decision to perform Caesarean section has resulted directly from the radiological report. We would draw especial attention to the type of case in which clinical examination shows a minor-to-moderate inlet disproportion but in which X-ray examination also shows severe lower strait disproportion. This type of case is fortunately rarely seen but if the condition is unsuspected its mismanagement may well lead to a major obstetric tragedy.

Of the 4 cases assessed as "correct", 2 had enforced Caesarean section after unsuccessful trial of labour, 1 had a Caesarean section with an impaction of the head at the brim for a 60-hour labour, the birthweight being 7 pounds 7 ounces. In the 4th case there was a very difficult forceps delivery of a baby weighing 5 pounds 5 ounces in fixed low arrest.

Of the 2 cases assessed as "incorrect", 1 had a normal delivery of a 4 pounds 3 ounces baby at the 34th week. The other had an uneventful normal delivery of a 6 pounds 8 ounces baby. The prediction

here can only be described as a mental aberration and if re-examined the cases would be put in Group C.

In future practice the majority of cases placed in Group E might well have elective Caesarean section or, with the minority, a trial of labour.

Observations on the Caesarean Sections, Forceps Deliveries and Trial Labours in the Survey.

An analysis of the special forms of clinical conduct of cases in terms of prediction groups is shown in Table III.

Table III.

Analysis of Caesarean Sections, Forceps Deliveries and Trial Labours in Terms of Prediction Groups.

Clinical conduct	Prediction group	Total	Not assessable	Correct	Forceps aided no disproportion	Incorrect
Caesarean section (49 cases)	A	10	10	—	—	—
	B	6	6	—	—	—
	C	6	4	2	—	—
	D	9	7	2	—	—
	E	18	15	3	—	—
Forceps delivery (39 cases)	A	17	1	—	15	1
	B	7	1	2	3	1
	C	7	1	6	—	—
	D	7	1	6	—	—
	E	1	—	1	—	—
Trial labour (50 cases)	A	22	1	20	1	—
	B	7	—	7	—	—
	C	9	—	6	—	3
	D	9	—	6	—	3
	E	3	—	3	—	—

The Caesarean Section Cases.

In the series of 300 cases, 49 patients were delivered by Caesarean section. In 42 of these cases, the prediction could not be tested (not assessable). There was a "correct" prediction in the remaining 7 cases. In 1 of these, which was not booked as a trial labour, Caesarean section was performed because of total inlet arrest (this had been placed in Group E). In the other 6 cases (2 in each Groups C, D and E) under trial labour conditions, Caesarean section was the enforced outcome because of a failure of spontaneous delivery.

Of the 49 cases, 10 had had previous Caesarean section (A, 3; B, 2; C, 2; D, 2; E, 1) and no doubt, at least in the A and B cases, the obstetrician was influenced in his decision to operate by the existence of a scar in the uterus, although the radiological evidence was such that normal delivery could have been expected.

The indications for Caesarean section in the 49 cases are set out below, under their respective groups, in Table IV.

Cases with Previous Caesarean Sections but no Repetition.

Whilst discussing the Caesarean section cases it is of interest to note that in

the series there were 10 patients on whom Caesarean section had been previously performed for reported disproportion, but who were not booked for repeat Caesarean section: 9 of these cases were placed in Group A, and 1 in Group C. The latter case showed a low arrest and had a simple forceps delivery of an 8 pounds 1 ounce baby. Of the 9 Group A cases, 6 had uneventful deliveries, the birth weights being 8.3, 7.14, 6.1, 6.9, 7.2 and 8.6; the other 3 cases showed uterine inertia and were delivered by forceps without difficulty of infants weighing 7.12, 8.3 and 8.10.

In Group B there were 7 cases: 1 was regarded as "not assessable" (forceps applied for persistent occipito-posterior). In 3 cases forceps were applied for reasons other than disproportion (2 with prolonged disproportion (7 for persistent occipito-posterior, 2 "low" forceps in heart cases, 4 for primary uterine inertia, 2 for foetal distress, 1 for maternal distress). In 3 of the cases previous Caesarean sections had been performed, the forceps being applied in each for uterine inertia. The remaining case, one of deep transverse arrest, was assessed as an "incorrect" prediction.

TABLE IV.

Indications for Caesarean Section Under Prediction Groups.

Indication	Case numbers in prediction groups					
	A	B	C	D	E	Total
Previous Caesarean	3	2	2	2	1	10
Disproportion in primigravida	–	–	–	2	13	15
Disproportion with history of previous difficult delivery	–	–	–	3	2	5
Failed trial labour	–	–	2	2	2	6
Elderly primigravida	4	–	–	–	–	4
Previous stillbirth after difficult delivery ...	–	1	2	–	–	3
Mitral stenosis	1	1	–	–	–	2
Placenta praevia	1	–	–	–	–	1
Miscellaneous	1	2	–	–	–	3
Group totals	10	6	6	9	18	49

The Forceps Cases.

A review of the forceps cases is of especial interest in relation to the forecast of difficulty at the lower pelvic levels—a difficulty by no means easy to anticipate clinically before the onset of labour.

There were 39 forceps deliveries in the series of 300 cases (Table V). In Group A there were 17 cases. In 16 of these the forceps were applied for reasons other than second stage and persistent occipito-posterior, 1 with Bonney forceps in a breech delivery). In 2 cases with deep transverse arrest the prediction was regarded as "correct" in view of the clear reservations made in the radiological report. One case was regarded as "incorrect."

Thus in Groups A and B, of the 24 forceps cases, 2 were "not assessable", 2 were "incorrect" predictions and in 20 the

predictions were "substantially correct" (i.e. 2 "correct" and 18 with forceps aid but no disproportion).

In Groups C, D and E there were 15 forceps deliveries: 2 cases were "not assessable." In the remaining 13 cases, forceps were applied for manifest disproportion during labour; thus in all of the 13 "assessable" cases, "correct" predictions were made.

TABLE V.

Indications for Forceps Application Under Prediction Groups.

Indication	Case numbers in prediction groups					
	A	B	C	D	E	Total
Persistent occipito-posterior	7	3	3	1	–	14
Mid-cavity arrest	–	–	–	2	–	2
Deep arrest	1	3	3	3	1	11
Primary uterine inertia	4	–	1	–	–	5
Foetal distress	2	–	–	–	–	2
Maternal distress	1	–	–	1	–	2
Heart case (low)	2	–	–	–	–	2
Breech (Bonney)	–	1	–	–	–	1
Group totals	17	7	7	7	1	39

From the clinical viewpoint, the engagement of the head in the pelvic brim before the onset of labour, or the ability to push the head well down into the pelvic cavity, are factors which enable the obstetrician, in the great majority of cases, to infer that the foetus will descend satisfactorily through the upper levels of the pelvis.

To anticipate difficulties at the lower pelvic levels by clinical methods is a much less easy matter when mid-cavity or low arrest is encountered, late and enforced Caesarean section carries a greatly increased risk. One can try to estimate the convergence of the side walls, the prominence of the ischial spines, the flatness and hollowing of the sacrum and the narrowing of the subpubic arch—but when all the findings are evaluated, the resulting information is not very precise.

If, by radiological study, forecasts can be made, with only few errors, of the likelihood of forceps delivery and of the ease, difficulty or danger of such delivery, a great service will be rendered to the obstetrician.

This series of forceps deliveries is too small to use as a basis for dogmatic conclusions, but it does suggest that adequate radiological investigation is of distinct value to the clinician in forecasting difficulties at the lower pelvic levels, so enabling him to avoid late Caesarean section or difficult forceps delivery with laceration and stillbirth.

The Trial Labours.

There were 50 trial labours in the survey of 300 cases. In Groups A and B there were 29 cases, in only 1 of which was there any dystocia; thus in 28 cases put down for trial labour on clinical grounds the radiological prediction of "normal delivery" was justified.

In Groups C and D there were 18 cases, 12 with "correct" predictions and 6 with "incorrect" predictions because normal delivery occurred without difficulty.

In Group E there were 3 cases, all with "correct" predictions: 2 of these had enforced Caesarean section and the third ended in a very difficult forceps delivery of a 5 pounds 5 ounces baby.

The Stillbirths.

In our survey there were 8 stillbirths, disposed in the prediction groups as follows: A, 4; B, 2; C, 1; D, 1; E, 0. In Group A, 2 were assessed as "correct" and 2 as "forceps-aid, no disproportion". The 4 cases in the other groups were assessed as "correct," of which 3 had forceps deliveries.

Thus, in 300 deliveries, there were 8 stillbirths, in 5 of which forceps were applied, one ending with craniotomy.

There was 1 maternal death, from acute eclampsia, in a Group B case with forceps application and stillbirth.

A Note on the Pelvic Types in the Persistent Occipito-Posterior Cases Requiring Forceps Deliveries.

Fourteen cases were delivered by forceps because of a persistent occipito-posterior position. The form of the inlet in these cases was:

Gynaecoid 6, android 2, android-pithecoid 1, pithecoid-android 2, pithecoid 3.

If the shape of the hind pelvis at the inlet plane alone is considered, it is found that the character of this pelvic zone was gynaecoid in 6 cases, android in 3 cases and pithecoid in 5 cases.

This brief note serves to emphasize the significance of pithecoid and android characters in promoting occipito-posterior positions. It is of interest to note that in only 1 of these 15 cases was there a posterior position of the occiput before the onset of labour.

A Note on the Problem of the High Head.

One of the commonest indications for pelvimetric examination is that "the head is high" or that "the head will not push in easily," the obstetrician being clearly suspicious of some inlet disproportion. From 1942 at Queen Charlotte's Hospital the lateral radiograph has been taken routinely with the patient standing and it is seen from such erect radiographs that in a large proportion of cases referred for "high head" that the head is, in fact, engaged. It is clear that examination in the erect posture often will show that no "high head" problem exists. It is now our practice, when the request form states that a "high head" is the indication for reference of the patient, firstly to take a lateral erect film only. If this shows the head to be engaging or engaged, the film is submitted to the obstetrician who is asked to decide whether a complete pelvimetric examination is necessary. It should be stressed that only cases upon which a complete examination has been made are included in this survey.

Surely an important elaboration of routine antenatal palpation is indicated by these findings. If the head is found to be high or difficult to push into the brim with the patient supine or sitting, then that patient should be examined standing. It is suggested that many a "high head" will then be found to have engaged, and that no real problem remains in such cases.

Conclusion.

A brief review of the overall figures in the survey will serve to conclude this paper.

(1) In the series of 222 cases delivered without any manifestation of dispropor-

tion, 200 cases (90 per cent) were predicted as likely to have delivery without disproportion.

(2) Of 54 cases having abnormal deliveries due to disproportion, 51 (94.4 per cent) were predicted as likely to have abnormal or difficult deliveries because of disproportion.

(3) Of the whole survey of 300 suspect cases referred by expert clinicians, the prediction was wholly "correct" in 83 per cent of the 253 assessable cases and "substantially correct" in 90.11 per cent; the difference between "wholly correct" and "substantially correct" being explained by the inclusion in the "substantially correct" figures of those cases with forceps-aided deliveries but without signs of disproportion during labour.

These figures may be regarded as a measure of the reliability of information which was obtained by radiological pelvimetry at Queen Charlotte's Hospital in a group of cases which had been selected mostly by experienced obstetricians because they presented some clinical antenatal problem—a critically selected group. It is suggested that with due interest on the part of the radiologist the prediction or prognosis of the course of delivery in terms of relative foetal-pelvic proportions, may reach a high degree of reliability, therefore it may be inferred that the examination and the opinion based upon it, may be of very great value.

In our series those predictions which have been proved to be erroneous have, almost without exception, been unduly pessimistic. Such errors have not once led to obstetric tragedy but rather have ensured that added watchfulness has been maintained in patients whose pelves could not be regarded as functionally ideal in shape or in size, qualitatively or quantitatively for childbearing.

We sincerely hope that this paper will give those obstetricians who are interested in the radiological study of foetal-pelvic relationships a clearer concept of the reliability of the method. Further, we hope that it will stimulate those radiologists who have not evinced any especial interest in obstetric radiology to prove to their obstetric colleagues, and to themselves, the considerable value of the information that may be gained.

Whilst radiological examination and opinion must essentially remain in a secondary place to clinical examination and opinion, we feel that antenatal X-ray studies should play an important role in helping obstetricians towards the achievement of an increasing accuracy in the antenatal assessment of the likely course of labour, and to aid them in their choice of the best method of delivery in borderline or difficult cases.

We trust that this survey will help towards this goal.

ACKNOWLEDGMENTS.

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Combined Anterior Pituitary Necrosis and Bilateral Cortical Necrosis of the Kidneys, Following Concealed Accidental Haemorrhage.*

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WE think that the following case of concealed accidental haemorrhage complicated by both pituitary and renal necrosis, the first to be described in this country, is worth recording. Analysis of the findings suggests various points which may throw some light on the mechanism of production of these complications.

CASE REPORT.

Mrs. E. C., aged 31, 2-para; was admitted to this hospital on August 27th, 1945, complaining of severe constant abdominal pain of 5 hours duration. Her past and family history were not significant. In 1937 she had had a full time normal delivery. In 1941 she had had a 7-months' premature stillbirth, attributed to the cord being round the child's neck.

History of present pregnancy. Last normal menstrual period, January 20th, 1945. Expected date of delivery October 27th, 1945. She booked at her local clinic on June 26th, 1945, and attended again on July 25th. The urine showed no abnormality on these 2 visits, and the blood-pressure readings were 100/70 and 100/60 respectively. The Wassermann and Kahn reactions were negative. Evidently she was not a co-operative patient since she did not take vitamins and iron as advised. Since the latter attendance she had had occasional frontal headaches without visual disturbances. Appetite and sleep were normal, but there had been some oedema of the feet for the 3 days prior to admission.

She was watching cricket on the afternoon of the 26th August, and on returning home felt a desire to micturate, went to the toilet and passed a small quantity of urine. Then it was, at 7.15 p.m., that she began to have severe, constant, abdominal pain. She felt "her tummy turn, tighten, and fill up." She fainted and was admitted to this hospital 5 hours later; having vomited several times, but having had no bleeding or shoulder pain.

On admission the patient was found to be very cold and shocked with marked pallor, oedema of the legs two-thirds of the way up to the knees and also of the lower abdominal wall; no icterus. The pulse was 120, of fair volume; blood-pressure, 150/80. Abdomen: Fundus, size of 34 weeks' gestation (31 weeks by dates), very tense and tender and foetal parts not palpable; foetal heart not heard. Fundi and discs normal. Vaginal inspection revealed very slight bleeding. A diagnosis of concealed accidental haemorrhage was made, and the patient immediately treated for shock on the usual lines of heat, blocks to the foot of the bed, injection of morphia, gr. ¼, and, after blood had been taken for a haemoglobin (5 g. per cent) and blood urea (57 mg. per cent) estimation, she was transfused with 2 pints of Group O_4 blood; there being no rhesus negative blood available at the time.

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Progress. While setting up the transfusion the membranes bulged at the perineum; these were artificially ruptured and non-blood-stained liquor escaped. Shortly afterwards a fresh stillborn infant with placenta and membranes were delivered together.

Twenty-six ounces of old and fresh clot and torn placental cotyledons were then expressed, and the patient was given an intramuscular injection of ergometrine 0.5 mg. There was very slight loss afterwards. The placenta was grossly irregular and indented throughout almost half its area, being 9 ounces in weight. Immediately after delivery a catheter specimen of urine, total ½ ounce, revealed 750 mg. albumin per cent.

Thereafter the patient was nursed in a darkened quiet room. She vomited on and off but managed to retain water. During the next night she passed very small quantities of urine on 2 occasions. On the morning of the 28th she was still oedematous, and also, now, were her face and eyelids. There was severe frontal headache, and she was still vomiting. The pulse was 92 per minute, blood-pressure 130/80, blood urea 106 mg. per cent. During the day a further 1 ounce of urine was passed, but as there appeared to be almost complete suppression of urine, she was given 1 litre of isotonic sodium lactate intravenously, in an attempt to bring about diuresis. This was followed by 1 litre of 5 per cent glucose saline at a slow drip.

Next morning, August 29th, only a few drops of urine were obtained by catheter, and there was definite bilateral renal tenderness. For the next 2 days her general condition remained unaltered, although by the graphs (Fig. 1) it will be seen that her output of urine was still negligible, and the blood urea and serum potassium were rising. On the morning of August 31st she complained of indigestion and bilateral renal pain. The tongue was moist and slightly coated, there was no alteration in the oedema, and the fundi and discs were normal. By the evening she was rather drowsy and the respirations somewhat acidotic, and she was not drinking well. She suddenly collapsed and died on the morning of September 1st, the 7th day of her illness.

At no time were measures required for the treatment of symptoms of engorged breasts.

Although the intake of fluid had been satisfactory until the last 18 hours, her total output of urine from the onset of abdominal pain had been approximately 4½ ounces in 5½ days.

The postmortem blood findings were: blood urea, 216 mg. per cent; serum potassium, 32.8 mg. per cent; plasma chlorides, 505 mg. per cent. In addition her blood group was found to be A2 Rh negative, with no anti-Rh agglutinins in her serum. The husband was group O4 Rh positive.

Postmortem Findings.

The postmortem examination was made 3 hours after death. The abdomen contained 150 c.cm. clear fluid, the right pleural space 350 c.cm. and the left space 150 c.cm. clear effusion. The tissues in general were oedematous.

Genito-urinary system. The right kidney weighed 210 g., the left 240 g. Perinephric fat was abundant. The capsules stripped easily revealing a smooth subcapsular surface mottled by alternating small haemorrhagic and intensely pale zones (Fig. 2). The cut surfaces showed a striking widespread anaemic infarction limited to the cortex and columns of Bertini (Fig. 2). There were bands 1 to 7 cm. long, wedges and small irregular areas a few mm. across of pinkish-yellow, firm, coagulative necrosis, bordered by a fine wavy margin of reactive hyperaemia about 1 mm. thick. These hyperaemic borders were both subcapsular and in the cortico-medullary demarcation line. Scattered between the infarcts were a few streaks of surviving cortex. The medulla was congested. All visible branches of the renal arteries and veins were free from thrombus. The renal pelves were normal, the left ureter normal, the right ureter slightly dilated. The bladder was markedly contracted, its mucosa oedematous, the ureteric orifices patent.

The uterus was enlarged and thick walled (3 to 4 cm. in its thickest portion) and showed 2 large crops of subperitoneal petechiae lying round the origin of each Fallopian tube and extending on to the Fallopian tubes and round ligaments. The cut surface showed extension of the petechiae into the superficial myometrium. Many venous sinusoids in the uterine wall were thrombosed. The placental site appeared healthy. There was a small atrophic corpus luteum of pregnancy in the left ovary. Neither ovary was engorged. The right ovarian vein was enormously distended by thrombus (maximum diameter 3 × 2.5 cm.) from its origin up to its entry into the inferior vena cava,

into the lumen of which clot protruded. But there was no main caval thrombosis. The proximal 8 cm. of the left ovarian vein was also thrombosed, but not distended.

was enlarged, unusually pale (rendering the pattern obscure) and showed scattered dilated subcapsular veins. The gallbladder contained about 20 facetted mixed stones, though its mucosa and duct

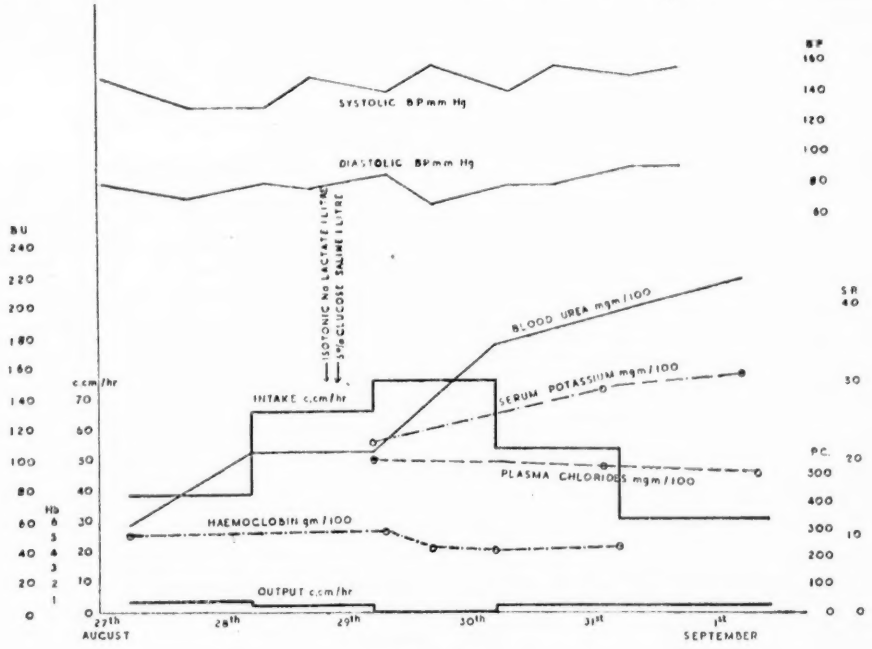

FIG. 1

CHART SHOWING CLINICAL AND BIOCHEMICAL FINDINGS.

Note from above down: 1. Blood pressure readings: note absence of hypotension. 2. Intake maintained and output negligible. 3. Biochemical findings (rising blood urea and serum potassium, and falling plasma chlorides). 4. Haemoglobin readings. Death occurred on the 6th morning.

Cardiovascular system. The valves, chambers and muscles of the heart (280 g.) appeared healthy. The coronary arteries, the aorta and branches were smooth walled, elastic and free from atheroma. There was postmortem clot in the pulmonary artery.

Respiratory system. Apart from a moderate degree of marginal emphysema and partial collapse of their bases, the lungs appeared healthy. The mediastinal lymph-nodes were normal.

Alimentary system. The tongue was moderately furred. The pharynx, oesophagus, stomach and intestines all appeared normal. The liver (1920 g.) appeared healthy. The bile-ducts were patent. The pancreas appeared healthy.

The spleen (300 g.) was enlarged, its pulp firm, the Malpighian bodies prominent. The adrenals appeared normal. The thyroid gland was rich in colloid. The breasts were heavy and tense and when incised gave out spurts of milk. The vertebral bone-marrow was pale. The right femur contained pink marrow in its upper two thirds, but did not show any gross medullary bone resorption. The brain (1280 g.) and its basal vessels were normal. There was a haemorrhagic oedema of the pituitary stalk. The hypothalamus was not engorged. The pituitary gland was fixed whole in

Zenker-formol solution and then bisected. After the chromate had been washed out the cut surface of the gland presented a massive anaemic infarction of the pars anterior bordered by a thin subcapsular haemorrhagic zone. The naked-eye appearance was identical with that of the renal coagulative necrosis (Fig. 2).

Microscopic Findings.

Pituitary. The anterior lobe shows a massive coagulative necrosis which has left only a few surviving cells scattered under the capsule and in the posterosuperior angle beneath and in front of the stalk. The pituitary cells have lost their haematoxyphil nuclear staining. The central sinusoids are empty but those nearer the periphery contain numerous polymorphonuclear leucocytes (many of them vacuolated and showing pyknotic nuclei) (Fig. 3). The sinusoids supplying the surviving cells are engorged. The basement-membranes can still be stained by aniline blue so as to show up the 'skeleton' of the infarcted area. And many of the parenchymatous cells, which stain a uniform pink with haematoxylin and eosin, show ghostly remnants of nuclei and granules when stained by Mallory's or Biggart's method. The pars intermedia and posterior appear normal. The sinusoids of the stalk are intensely engorged and the interstitial tissue at its upper end is flooded with red cells. The arterioles are not thrombosed. But the veins of the stalk, both large and small, contain varying types of thrombus: amorphous hyalinized red-cell masses, organizing fibrin and entangled cells, and conglutinated red cells (Fig. 4). The age of the pars anterior necrosis, judged by the parenchymatous karyolysis, the polymorphonuclear infiltration and the absence of secondary atrophy lies between 3 and 9 days and tallies with the 6 days' survival after delivery.

Kidneys. The cortex and columns of Bertini show a widespread ischaemic coagulative necrosis split up by slender streaks of surviving renal tissue which is also present as a thin layer underneath the capsule (Fig. 5). The medulla is congested but nowhere infarcted. In the depths of the infarcted cortex, karolysis is complete and there is no leucocytic infiltration; the ghosts of glomeruli, tubules and vessels (including dead interlobular arteries distended with hyalinized fibrin thrombus) are readily made out (Fig. 6). The edges of the infarcted areas show a heavy interstitial infiltration with degenerate fatty polymorphonuclears and occasional patches of interstitial haemorrhage. The zone of demarcation between living and dead cortex is surprisingly sharp. It cuts right across the renal structures so as to leave some arterioles and some glomeruli bisected into a living and a dead half. There is not any evidence in the living glomeruli of any previous widespread ischaemic changes nor of any eclamptic oedema of the capillary basement membranes. The living tubules contain cast material and desquamated cells, a few of the living cells are fatty. There are occasional terminal interlobular and afferent arterioles in the living cortex which are distended and occluded by hyalinized material (resembling fused red cells). These vessels are surrounded by a small zone of polymorphonuclears and histiocytes. Elastic stains confirm the extraordinary, almost "paralytic" dilation of some of the interlobular arteries, particularly in the infarcts (Fig. 6). These stand out in all sections. Non-thrombosed interlobular arteries at the edge of the infarcted tissue show commencing acute necrosis of their walls and a surrounding zone of radially grouped pyknotic leucocytes—a typical acute arteritis (Fig. 7). The interlobular and arcuate arteries and veins are free from thrombus or disease, some interlobular arteries show a mild elastosis. The age of the infarcts, judged by the degree of parenchymatous karyolysis and marginal interstitial polymorphonuclear infiltration, tallies well with the clinical history of 6 days anuria. Thus, the renal necrosis judged histologically is likely to be of the same age as the pituitary necrosis.

The picture as a whole suggests the following possible interpretation of the train of events. The infarcted terrain is that supplied by the interlobular arteries. These appear to have been primarily obstructed by stasis leading to thrombosis which has spread both backwards and forwards along these vessels. The grossly diminished or complete loss of blood-flow led to renal functional failure and ischaemic necrosis. How the stasis arose and the significance of the arteritis are discussed later.

Pancreas. The glands and islets are normal. But there is a striking arteritis in all stages of development which involves many of the small and large vessels. The earlier lesions consist of fibrinoid

necrosis of a segment of the adventitial coat. The necrotic collagen is infiltrated with pyknotic nuclei. The next development seen is an extension of the necrosis into the media and a brisk perivascular inflammation. In the advanced stages, the whole of the adventitia and much of the media is swollen and replaced by fibrinoid material and somewhat radially arranged pyknotic nuclei (Fig. 8). The perivascular interstitial tissue is heavily infiltrated with histiocytes and polymorphonuclears some of which extend into the surrounding pancreatic glandular tissue. The lumen of the vessels is free from thrombus. The only thrombosed vessels present are a few small arterioles and venules lying in the neighbourhood of a gravely involved larger vessel.

Liver. There is a widespread fine fatty droplet change affecting all zones of the lobules and, in addition, occasional centrilobular focal necroses (small collections of dead liver cells infiltrated with fresh polymorphonuclears). There is no eclamptic periportal haemorrhagic necrosis.

Uterine wall. Some of the subperitoneal muscle fibres are vacuolated and show glycogenic degeneration of their nuclei. This same layer is rendered prominent by the presence of numerous interstitial haemorrhages. Many uterine veins are distended and filled with aseptic fibrin thrombus showing, as yet, practically no organization. The uterus is lined by fibrin, entangled red cells and masses of pyknotic nuclei derived from decidual cells and polymorphonuclears. There are scattered cystic healthy endometrial glands of the basalis layer.

Right ovarian vein. This is distended by thrombus which consists chiefly of massed red cells (still containing haemoglobin) traversed by strands of fibrin, conglomerated platelets and entangled leucocytes. It all looks of recent formation (less than 5 days). The muscularis has been thinned by distension. The adventitia is oedematous and contains fibrin and free red cells which have spread a little way into the surrounding fatty connective-tissue.

Spleen. There is a moderate increase of polymorphonuclears in the pulp and occasional megakaryocytes in the sinusoids.

Gall bladder. The outer connective-tissue coat shows a mild perivascular inflammatory infiltration with lymphocytes and neutrophil and eosinophil polymorphonuclear leucocytes.

Breast. The acini show a well-marked lactation hyperplasia. Their lumens and lining cells are distended with fat droplets.

Sections of the lung, heart-muscle, hypothalumus, bone-marrow, cervix, adrenal, thyroid and left ovary (which contains a well vascularized corpus luteum of advanced pregnancy) do not show any essential pathological changes.

It is clear from the above clinical history and the naked-eye and microscopic postmortem findings that death was due to renal failure brought on by the massive renal necrosis which had most likely occurred 6 days previously at the time of the concealed accidental haemorrhage. One cannot gauge to what extent the pituitary necrosis, which had occurred at the same time, contributed towards the death.

Discussion.

Sheehan[1] found 11 cases of postpartum necrosis of the anterior pituitary in a consecutive series of 59 postmortem examinations of women dying in the puerperium. Thus, there is about a 1 in 5 chance of its occurrence together with renal cortical necrosis. He also found that in 24 of 28 recorded cases there was a history of haemorrhage at delivery. All these patients were "collapsed." Since haemorrhage and "collapse" are present in almost all cases of renal cortical necrosis, the chances of its coincidence with pituitary necrosis must be even higher than 1 in 5. Sheehan thought the pituitary necrosis resulted from haemorrhage and "collapse" rather than "toxaemia."

Tomlinson[2] described a case of combined pituitary and renal necrosis in a multipara aged 28, who was admitted in the 38th week of her third pregnancy suffering from a urinary tract infection. Following forceps delivery of a stillborn infant and manual removal of the placenta she went into severe shock. She developed anuria and died 6 days later of uraemia. Sheldon and Hertig[3] described 2 cases of renal cortical necrosis in which postmortem examination

FIG. 2.

Right kidney and pituitary. (Photograph reduced to half natural size.) Widespread pale cortical necrosis bordered by a thin hyperaemic zone, mottled subcapsular surface, ischaemic cut surface of pituitary gland.

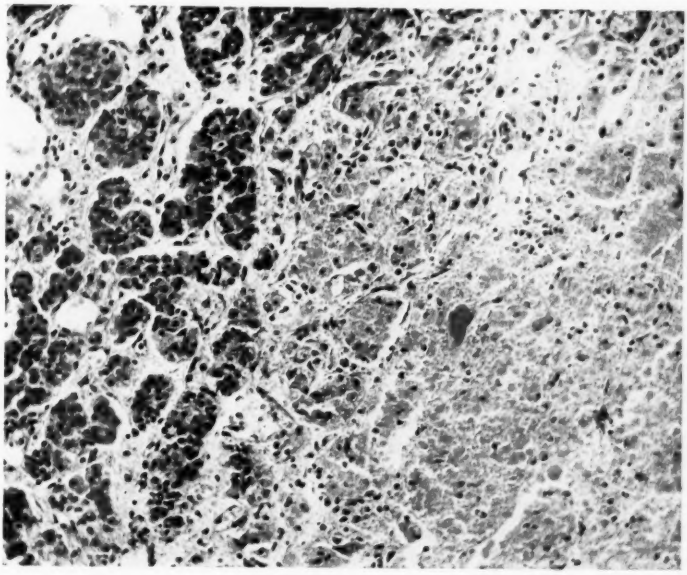

FIG. 3.

Pituitary gland. (Photomicrograph [× 175], Formol-Zenker, Haematoxylin and Eosin.) Border between living cells of the pars anterior to the left and dead to the right.

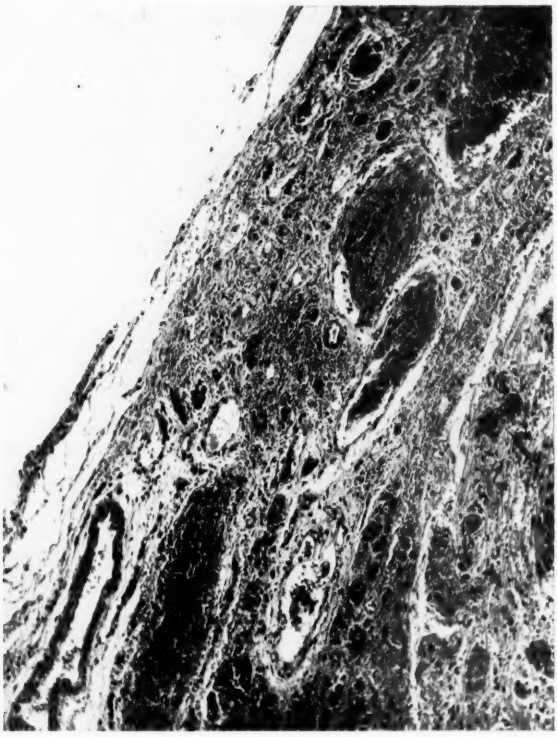

FIG. 4.

Pituitary stalk. (Photomicrograph [× 90], Formol-Zenker, Haematoxylin and Eosin.) Thrombosed veins in the pituitary stalk. The arteries and arterioles are patent.

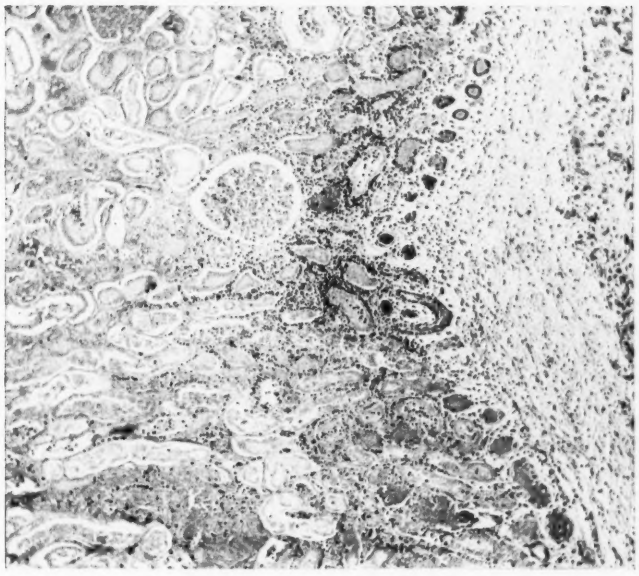

FIG. 5.

Kidney. (Photomicrograph [× 90], Formol-Zenker, Haematoxylin and Eosin.) Oedematous congested renal capsule separated by a thin hyperaemic zone infiltrated with leucocytes from underlying infarcted cortex.

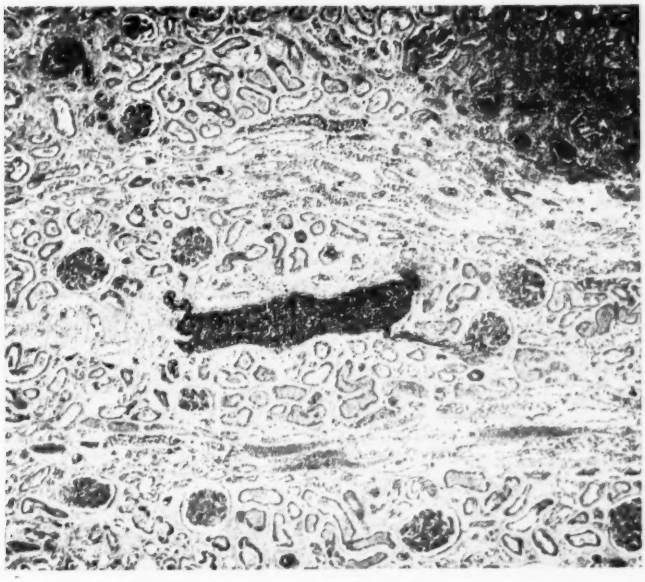

FIG. 6.

Kidney. (Photomicrograph [× 60], Formol-Zenker, Haematoxylin and Eosin.) Distended thrombosed interlobular artery giving off two afferent arterioles which are also thrombosed, in the infarcted cortex.

FIG. 7.

Kidney. (Photomicrograph [× 175], Formol-Zenker, Haematoxylin and Eosin.) Acute arteritis of an interlobular artery at the edge of an infarcted area.

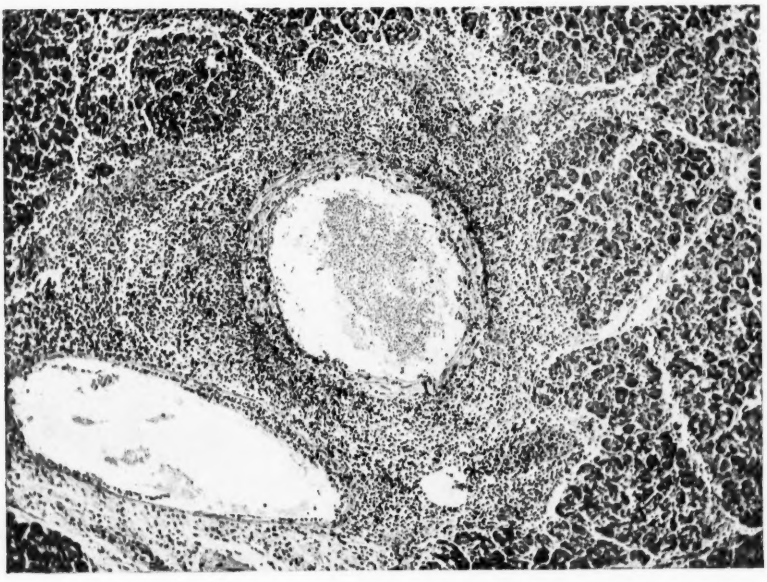

FIG. 8.

Pancreas. (Photomicrograph [× 90], Formol-Zenker, Haematoxylin and Eosin.) Acute necrotizing arteritis with inflammation spreading into the interstitial tissue.

I.D. & A.H.C.W.

revealed anterior pituitary necrosis as well. The 1st was a multipara aged 33 who went into labour 7 weeks from term following signs of toxic separation of the placenta. She died 7 days later having passed only 30 c.cm. of urine since delivery. The 2nd was a multipara, aged 40, who went into labour 8 weeks from term following signs of toxic separation of the placenta. She died 11 days later, having passed 53 c.cm. urine since delivery.

Duff and More[4] reviewed the literature up to date on symmetrical cortical renal necrosis. They found a total of 48 authentic cases following pregnancy. Pituitary necrosis was not described in any of these. But, after reading the case histories, one is inclined to believe that in very few, if any, was the pituitary gland examined microscopically. Since 1941, 6 cases of renal necrosis have been described and an associated pituitary necrosis found in 3 of them, as quoted above. It seems reasonable to presume that a careful examination in future cases of renal necrosis will reveal a frequent association with pituitary necrosis, and that our case, and those of Tomlinson, Sheldon and Hertig, are by no means the rarities they at present appear to be.

Each of these 2 conditions has been excellently reviewed[1, 4] already, but various points in our own case are worth further discussion.

Pituitary gland. The haemorrhagic state of the stalk and the presence in it of thrombosed sinusoids were also seen by Sheldon and Hertig. The anatomy of the venous sinusoids of the stalk has been carefully investigated by Wisloki and King.[5] They proved that these are "portal" veins which supply the sinusoids of the pars anterior together with branches of the superior hypophyseal arteries. The sinusoids of the pars anterior drain into the cavernous sinus. Popa and Fielding,[6] who first identified the pituitary "portal" veins, believed, on the contrary, that the direction of flow was towards the hypothalamus. The findings in this case of a necrotic pars anterior and a normal hypothalamus confirm Wislocki's view of the direction of flow, and at the same time imply that the pituitary "portal" veins play a much bigger part than the arteries in the blood supply of the pars anterior, at all events, in late pregnancy.

Accidental haemorrhage. We could not find a cause for the retroplacental haemorrhage. The histology of the uterus ruled out sepsis. There was not any evidence of eclampsia or essential hypertension or glomerulonephritis in the kidneys. The section of right ovarian vein showed the thrombosis to be of too recent origin to have played any precipitating role.

Renal necrosis. It is possible to suggest a vascular mechanism without resort to toxins. Duff and More[4] concluded in their review that "some unknown factors, given suitable conditions, cause disturbances in the terminal arteries of the renal cortex, the damage done depending on the sensitivity of these vessels to irritation. That in pregnancy these vessels may become hypersensitive. That varying hypersensitivity and intensity of irritation may lead to a series of vascular degenerative changes, including necrosis of the wall and thrombosis. That the same sized arteries of other organs are also hypersensitive; though to a less degree." It seems to us that the unknown factor may be simply the reflex vasoconstriction which follows blood loss and is directed towards accommodating the vessels to the reduced blood volume and maintenance of the mean arterial pressure. The point that there is a definite association of symmetrical renal necrosis with haemorrhage and collapse needs no labouring.

Evidence for a reflex vasoconstriction can be seen that among Duff and More's 48 cases of renal necrosis, in the 21 which followed accidental haemorrhage, the average blood-pressure was 170/109 (the lowest recorded was 118/50).

In their studies on the renal circulation in shock Lauson, Bradley and Cournand[7] found evidence of a considerable degree of renal vasoconstriction in most cases (including the post-haemorrhagic state), and indications that blood is shunted away from the kidneys. (thus confirming the generally held view that the oliguria or anuria of shock results from decreased circulation through the kidneys). We have some clinical evidence in our own case that there was a generalized vasoconstriction following the 26 ounces retroplacental haemorrhage in that the patient was "shocked," pale, anuric and maintained a blood-pressure of 150/80. Granted the hypersensitivity of the terminal renal arteries in pregnancy, renal arteriolar spasm would undoubtedly follow. Whereas relief of the spasm is followed normally by continuation of the circulation at a reasonable renal filtration pressure, in this case there appears to have been a block due to massed red cell thrombus. The possible factors responsible for this might have been intensity of the vasoconstriction, or a paralytic vascular dilatation with stasis following the spasm (Scriver and Oertel[8]) or the increased coagulability of the blood said to occur in the postpartum state. Outside the kidneys, the centrilobular hepatic focal necroses are further evidence of a reduced blood flow as found by Bywaters[9] in his studies of the liver in crushing injuries. It is interesting to note that the degree of anaemia 5 g. per cent haemoglobin, would further aggravate the ischaemic state of the kidneys and other sensitive viscera. In the 9 cases of renal necrosis in the literature in which the haemoglobin level was given, the average figure was 6 g. per cent. This level, after discounting the "physiological" low normal value in late pregnancy and the drop due to haemorrhage, suggests that these patients were anaemic before their catastrophe.

Arteritis. Apart from haemorrhages in the heart, brain and stomach, foci of necrosis round vessels in the pancreas,[4] and in the caecum and adrenals[3] have been described. They have been attributed either to vascular spasm or to the action of some toxic substance. The pancreatic arteritis in our case (as well as the renal) appeared definitely to have commenced in the adventitia. The age of the lesion, judged by the freshness of the polymorphonuclears bordering the necrotic areas, appears on the whole to be definitely less rather than more than 6 days. It is conceivable that it is an ischaemic necrosis resulting from diminished blood flow through the vasa vasorum associated with spasm both of the vasa and the vessel itself. The uraemic state of the patient might account for any "toxic" element in the aetiology of the arteritis. It is relevant to recall that Byrom[10] produced renal cortical necrosis and a necrotizing arteritis of the renal and gastric arteries of rats by repeated subcutaneous injections of vasopressin.

Any factor which causes complete obstruction to the blood flow through the terminal renal arteries will produce a similar ultimate pathological picture of symmetrical renal coagulative necrosis. And since not all cases of such renal necrosis are associated with haemorrhage, or for that matter with pregnancy, there must be more than one mechanism which obstructs the renal circulation. Similarly, arteritis can result from other causes than spasm (for example following sulphonamide hypersensitivity).

But the reflex vasoconstrictive mechanism

described above seems to us to fit in well with the typical findings in cases associated with accidental haemorrhage.

Conclusion.

We suggest that, as in the case of pituitary necrosis, pregnancy renders the patient liable to suffer symmetrical cortical necrosis as a complication of haemorrhage and collapse. Though the latter is a less frequent development than the former, it is not surprising to find the two conditions in common association. The altered circulatory state produces spasm and thrombosis of the renal terminal arterial segments, stasis and thrombosis of the stalk segment of the pituitary "portal" venous system. The resultant anoxia of the tissues supplied by these vessels may be aggravated by the anaemia associated with many of these cases. One may find further evidence of anoxic necrosis in the liver and possibly in the walls of small arteries (in the pancreas and adrenals).

Acknowledgments.

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Infertility in Women

BY

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THE subfertility problem is at long last being tackled with considerable zest in this country, as is evidenced by clinics that are springing up, manned by enthusiastic personnel. This being so, it may be suitable at this stage to pause for a while in order to consider the present position as regards the investigation of this complex subject.

Just now it looks as if there might develop a growing danger in some quarters of a tendency to over-elaboration of technique, for it would seem that the bulk of the people of this country, whether urban or rural, is not ripe for as thorough investigations as those undertaken by our transatlantic friends; nor can the majority of them pack their suitcases and travel to centres where the "minimal sterility survey", as for example that drawn up by E. C. Hamblen, occupies at least 3 whole days. Again, it may be asked, is success always eugenically expedient in many of these stubbornly subfertile couples, when quality not quantity is surely the desired goal."

The investigation of subfertility falls into 3 main hands:

A. The family practitioner.

B. (1) The smaller of the provincial clinics, such as those sponsored by the Family Planning Association. The outshining example of these is one that has been run since 1931 by Dr. Margaret Jackson,[1] with the collaboration, as biologist, of Mrs. Clara Harvey, B.Sc.

B. (2) The ultra-specialized clinic, which can call on the services of a gynaecologist, andrologist, biochemist, biologist, pathologist, radiologist, endocrinologist, and psychiatrist, all working together.

It is to these clinics that we should look in the future for the regular publication of their research work and of their data for it is only by the pooling of all available results that we may hope to attain success.

(A) THE FAMILY PRACTITIONER.

Some subfertile couples are somewhat shy of asking their practitioner's advice on their problem and prefer to go straight to a clinic for investigation. Such a clinic rightly is always anxious to refer these cases back to their doctor for subsequent treatment, injections, etc.

Other couples realize that no one understands their home life and their habits better than their family medical adviser, and so they tend to ask his help, and frequently sooner in their married life than those who go straight to the clinic. If the doctor has the time and the assiduity, it is surprising how many of his attending subfertile couples he can handle with success, without ever referring them to a specialized clinic. By the time he has taken a careful history, carried out a thorough medical overhaul and a local examination of both partners, he will probably have encountered several subfertility factors, which he can set about correcting.

Frequently some minor gynaecological condition calls for suitable adjustment: or, again, advice about the correct time for coitus, the taking of the daily rectal waking

temperature, with an explanation of the preluteal drop, may be the next step. The carefully judged administration of thyroid in certain cases, or of dienoestrol with ethisterone, or of the gonadotrophic hormones in others, will call for a working knowledge of the sex-hormones. Possibly the suggesting of a glucose saline precoital douche may be followed by success or, if not, he may then be able to carry out for himself a Huhner's or Sims' post-coital test, followed by a rough sperm count. At this stage the practitioner may decide that an endometrial biopsy or a tubal insufflation or a hysterosalpingogram is indicated. This is when he will probably call in the clinic's help, if he has one to turn to in the neighbourhood.

(B) The Subfertility Clinic.

If the problem, however, is a more complex one, then it is usual to carry out a far more exhaustive investigation, depending on the facilities available at that particular clinic. The co-ordination and interpretation of the results of these examinations usually locates one or more subfertility factors among the following groups:

I. Pelvic pathological lesions, either inflammatory or neoplastic (the surgical treatment of these conditions will not be discussed in this paper).

II. The cervical factor—the nature of the cervical plug of mucus and the reception of the inseminated spermatozoa.

III. The tubal factor—possibly a case of lack of function; of occlusion; or of spasm.

IV. The endocrine aspect on the female side, with its varying effects on the reproductive function.

It should be possible to carry out the following special investigations at the clinic:

I. Examination of the vaginal fluid, and the possible taking of vaginal smears.

II. Tubal insufflation.

III. Hysterosalpingography.

IV. Endometrial biopsy.

V. Post-coital test, whether after Huhner's or Sims' methods.

VI. Seminal analysis.

VII. The investigation of endocrine dysfunction, hormone analysis, etc.

I. The *examination of the vaginal fluid* is briefly with the object of excluding trichomonas vaginalis, or monilia, and with finding the *p*H value.

The vaginal smear—this diagnostic method has not, so far, been employed as extensively in this country as in America, where the technique of Papanicolaou and Shorr is usually followed.[2] The smears may be taken right through the cycle, showing a varying cytology and a rhythmic cyclicity in the various phases.

i. Menstrual phase smear.

ii. Postmenstrual (early proliferative) smear, 4–5 days.

iii. Pre-ovulation smear (late proliferative), 11–12 days.

iv. Ovulatory type smear, 13–14 days.

v. Post-ovulatory smear, 17-19 days.

vi. Premenstrual or secretory phase, from 19th day onwards.

From comparison of the above, help is claimed in estimating ovarian activity, in assessing the effectiveness of oestrogenic, androgenic, and gonadotrophic preparations, and in determining ovulation.

II. *Tubal insufflation*—Anyone who has seen a case of pelvic peritonitis following either insufflation or hysterosalpingography, will proceed very carefully in every case, and more so in any suspect case. Malpas considers "in the presence of inflammatory appendage disease or its sequelae neither procedure should be carried out except after very serious consideration, and then only if a vigorous bimanual examination and a course of short

wave pelvic diathermy have demonstrated complete quiescence".[3] The test is best performed either between the 4th and 10th day after the end of the period, or in the premenstrual phase, when a biopsy of the endometrium can be taken at the same time. The findings at insufflation are:

i. Normal patency—the pressure reaches 60 to 120 mm.Hg. and then rapidly falls.

ii. Non-patency.

iii. Spasm—the escape pressure is high, the flow does not take place at once but, when it does so, its fall is rapid. If the test is repeated later, normal patency is shown.

iv. Stenosis—the escape pressure is high, the fall is slow, and repetition of the test gives a similar result. If a kymograph machine is being used, which is the best as the peristalsis of tubal function is observed, then the tracing will be according to the above.

Sharman in his series of nearly 500 cases found several erroneous results.[4] In 60 non-patent to insufflation he found 6 patent to hysterosalpingography. So, in repeated insufflation cases, he drew the following conclusions:

i. There was not any material change in the tracing of a patient insufflated on 20 consecutive days (menstruation intervening).

ii. Anaesthesia had no effect on the tracing.

iii. Around ovulation time there is sometimes more active or more frequent peristalsis.

iv. No great variance in the tracing in repeat insufflations is found, even after several months, either in the level at which patency is established, or in the appearance of the tubal contraction-waves.

v. Repeated insufflations have also shown that spasm, whether of lesser or major degree, is not a constant state and that many tracings may show a normal patency.

vi. The result of a single insufflation, with or without anaesthesia, showing apparent non-patency, cannot be depended upon.

vii. As a therapeutic measure it was found that, of the ensuing pregnancies, 36 per cent became pregnant in 3 months and 50 per cent became pregnant in 6 months.

III. *Hysterosalpingography*. Where an X-ray apparatus is handy, and the extra expense is not a drawback, there is no doubt that a higher percentage of cases subsequently become pregnant after hysterosalpingography than after tubal insufflation. The test is usually done 4 to 10 days after the end of the period. The use of anaesthesia is still debatable—many prefer atropine or morphine, $\frac{3}{4}$ of an hour beforehand, without anaesthesia. There are many types of instruments, each gynaecologist usually keeping to one pattern. Neohydriol is probably the best type of oil, care being taken not to use too much pressure or too much oil. It is always a help to use the screen first—watching the oil passing along the tubes, usually after 5 to 6 c.cm. have been slowly injected. Sometimes there is intense spasm, when nothing passes through the sphincter muscles at the cornua. Many and various are the findings by this technique, with suitable treatment being called for of salpingography, intrauterine tubal implantation, shortening of the round ligaments, myomectomy—especially if the fibroid is submucous in type—etc. From the above considerations the accurate interpretation of a hysterosalpingogram may be difficult. It may show blockage when none exists, or its interpretation may be so difficult that an accurate conclusion cannot be reached. On the other hand it may show patency when insufflation has indicated blockage, and, in non-patent

tubes it will reveal the precise site of blockage. As Malpas says "both procedures, insufflation and salpingography, are complementary the one to the other and essentially diagnostic. Their use as primary therapeutic measures is likely to lead to many disappointments, and will tend to bring them into discredit. In general it may be said that, if the tubes are demonstrated patent, conception will ultimately occur in about half the cases. If careful steps can be taken to exclude the other causes of sterility—azoospermia, anovulatory cycles, endometrial tuberculosis—then the proportion of pregnancies following positive tests will very likely be still higher." [3]

IV. *Endometrial biopsy*. This is carried out in the premenstrual phase, usually between the 24th and 26th days of a 28 day cycle, with a Sharman curette, when there is no need for anaesthesia; but it is a procedure that should be done with the utmost gentleness. It is impossible to discuss the findings here, but briefly it may be said that it is rare to find an endometrium which shows no sign of progestational change, though in many this appears to be inadequate or incomplete. It would seem that the endometrial biopsy does not provide a complete answer to the question as to whether normal ovulation has occurred, though it does provide important information about ovarian function and about endometrial response. Occasionally no period has followed the taking of a biopsy, presumably after fertilization has occurred. Endometrial biopsy not only does not interfere with pregnancy but apparently encourages decidual formation. In Sharman's recent series, 335 showed the normal endometrial appearances of the premenstrual or post-ovulatory stage, and 23 exhibited definite anovular cycles.[4] Of these 23, none became pregnant during the period of study, and only 2 under treatment. Anovular menstruation is therefore a major infertility factor, when it occurs, although its incidence is low.

V. *The Post-coital test*. Recently Rubin has stated "our results in combating childlessness may be expected to improve the more we are prepared to investigate the causes of the infertile mating, rather than the infertile individual". This wise dictum, confirmed by so many, has tended to make some workers focus a great deal more of their attention on the cervix uteri and its secretion, the plug of cervical mucus. Barton and Wiesner[5,6] have considerably developed their technique of the Sims post-coital test, basing their work on a paper of Marion Sims of 1868. They describe the "ovulatory phase" cervical mucus plug as "a cordon of mucus which clings in a fairly well delineated cascade to the surface of the cervix. It is perfectly clear, colourless, rather viscous, and it has a low vapour pressure so that it can be examined at leisure without change by rapid drying, and it is invariably alkaline." They proceed to describe the cyclical changes in the appearance of the cervical mucus, with changes in the volume, the nature, and the opacity of the secretion, with the density of the sloughed cervical cells and of the leucocytes, in the second phase of the menstrual cycle, which may become of diagnostic importance, as a mirror of ovarian function. From research work on the hyperfertile male we may look for more help and guidance in the future. There can be few more interesting phenomena to watch through the microscope than when, under the centre of a vaseline-ringed cover slip, has been placed a drop of ovulatory cervical mucus. Then through a gap in the ring is placed a drop of semen, first on one side from one subject, then on another side from another subject, through another gap. Barton and Wiesner have described in detail their observations

of the cervical mucus, concluding that "first, spermatozoa penetrate into the mucus of their own power and motility. This penetration is attempted by many but achieved by only a comparatively small proportion of the sperms approaching the interlace. This again corresponds to observations *in vivo* where the total number of sperms penetrating into the cervix does not exceed a small proportion of the ejaculate. Thirdly, the spermatozoa which have penetrated into the mucus live longer than any of them do in the seminal fluid under comparable conditions". In summing up they are of the opinion that the cervix does not function as a mere passage or in a crude mechanical way, but as a highly developed filter and preserver for those sperms which satisfy certain stringent conditions. It is the secretion of the cervical mucus that is of paramount importance, depending on oestrogenic secretion for its presence and healthiness, quite apart from local pathological conditions, such as endocervicitis, which much impedes or inhibits cervical invasion. It is when the cervical mucus is normal, or nearly normal, and sperm invasion still fails that artificial cervical insemination may be considered. First, the husband's cells are treated to improve motility, and then, provided they show a reasonable degree of initial motility when collected—either directly or from the vaginal pool—there is carried out a deposition within the cervical canal of a small quantity of the husband's semen, in order to secure better invasion. This deposition of the husband's semen may be indicated apart from the above impaired cervical invasion by spermatozoa:

i. Where treatment of impotence has failed.

ii. Where the husband fails to ejaculate during intercourse or emits only a minute quantity of azoospermic liquid.

iii. In certain cases of incurable dyspareunia.

VI. *Seminal analysis* will be considered in the male section of this paper.

VII. *The investigation of endocrine dysfunction.* So vast has become the literature on the hormonal side of subfertility that even the briefest sketch of the problem and its treatment does not fall within the scope of this short sketch. It may be said that, as regards treatment, some of the newer preparations have begun to give better results. For example the microcrystalline suspension of oestrogenic hormones produces its action in a more gradual and sustained manner.

Again the combination of chorionic gonadotrophin and anterior pituitary-like hormone, in the compound synapoidin has proved useful.

Irradiation therapy, both of the pituitary and ovaries, as a supplement to hormone treatment, has been more advocated in America than anywhere. As regards the ovarian treatment, the Kaplan technique has met with some success in severe cases of amenorrhoea. In this country Green-Armytage and Carter Braine have reported a series of 7 cases with amenorrhoea up to 16 months in duration, 4 of whom conceived, and went to term, producing healthy babies.[7]

Among many other aspects of the endocrine side of the problem, the relation between fecundity and body temperature has come to be recognized as an important factor in dating ovulation and so deciding the optimum time for coitus. Van de Velde (in 1904 and 1929) showed that in the normal woman the body temperature is relatively low during the first and relatively high during the second half of the menstrual cycle. In this connexion Raymond Greene and Nieburgs, summarizing their findings,

said: "The temperature variations during the menstrual cycle closely correspond to changes in glandular activity, particularly of the pituitary, gonadotrophic, follicular and luteal hormones. It is suggested that the decrease of temperature is an expression of oestrogenic activity, where temperature rise is caused by the corpus luteum. Usually on the 14th day before menstruation oestrogen excretion reaches its highest peak, causing the sudden drop in temperature. This is followed by a vigorous release of luteinizing hormone from the anterior pituitary, which produces ovulation and corpus luteum formation, with increased progestin excretion. During the luteal phase the temperature rises to a level between 99° and 100°F. or more and remains at this level with slight fluctuations throughout the whole luteal phase. It declines 1 or 2 days before the onset of the menses."[8]

From this very superficial sketch of a very important national problem will be seen the crying need for accurate diagnosis, which will surely result from patient research into its many sub-sections, to mention only ovulation, the determination of faulty oögenesis and its promise of correction by hormonal administration, the biology of the cervix uteri, the whole question of hormonal analysis, and the bearing of the minor endocrinopathies and their suitable therapeutic adjustment.

In passing it might be wise to reflect on the conclusions of the Peckham Health Centre in their recent "Observations on the population question":[9]

1. That "parenthood", that is to say, biological growth and differentiation through the family, is a natural process with powerful urges operating through both male and female, but only in health.

2. That deep and primary biological causes underlie the frustration of parenthood.

3. That treatment of symptoms will not eradicate the cause of the symptoms, and so lead to cure of the disorder.

4. That, so far, all administration pertaining to parenthood—antenatal care, provision for confinement, child welfare, etc., all dealt with as seperate items—has been approached from the negative point of view of correction—the medical approach. If parenthood is to appear to the public as desirable, it must be approached from the positive point of view of cultivation, the biological approach.

There is a great deal of common sense behind these observations; there is much to be learned by combined research and sound team work in the newly organized clinics, and there will have to be a nation-wide propaganda campaign inaugurated, with the Government deciding how best maternity can be both encouraged and honoured and made practicable.

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Male Infertility

BY

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THE necessity for an examination of the husband, when the wife has failed to conceive, is now widely recognized, and it is no longer necessary to stress this point. There are no statistics by which the incidence of male fertility in this country can be assessed, but it is probable that in one-fifth of all barren unions the husband is mainly responsible and that in about two-thirds of them his infertility is a contributory factor. In the majority of cases the clinical examination does not reveal abnormality, and the true state of affairs is only discovered when the semen is analysed. Cases in which such obvious and gross lesions as hypogonadism, hypopituitarism, retained testis and induration of the epididymes are found are in the minority, and it is therefore never possible to assure a patient that he is fertile after the preliminary examination alone. This assurance can only be given subsequent to the semen analysis.

The preliminary investigation must not be confined to the genital tract, since lesions in other parts of the body may have repercussions on the germinal epithelium. A careful history must be taken of all past illnesses, and particularly of gross infections in recent years. The occupation of the patient should also be noted, because it is likely that some occupations which entail exposure to the action of various chemicals, or to electrical rays and excessive heat are dangerous to fertility. The mode of life and habits of the patient must also be noted.

Because conception depends on the presence of spermatozoa in the female passages at the time of ovulation, the husband should be questioned as to the frequency and effectiveness of sexual intercourse. Poor potency and infrequent intercourse is a contributory factor in many barren marriages, for if sexual union takes place on an average only once a month the chances of its coinciding with ovulation are considerably reduced. Unfortunately, poor potency is extremely common in this country and many of the husbands who are under treatment for reduced fertility also need help for sexual difficulties. Sometimes the latter is the direct result of the former. When a man's fertility is questioned he often develops the fear that there may also be something wrong with his virility, and as a result his sexual difficulties are increased. Premature ejaculation is perhaps the commonest of these difficulties, but although it reduces the likelihood of conception, it does not prevent it. It is important to make certain that the sexual act is completed, for absence of emission is by no means uncommon. Sometimes the patient himself is completely unaware of the fact that he has no emission, and it is only revealed after he has been asked to obtain semen for analysis. This grave defect is not due to the presence of any physical lesion but to some sexual aberration. In a group of 35 cases of this type analysis revealed 10 cases of severe sexual perversions, including 3 cases of homosexuality and 3 of sadomasochism. Only 4 of them responded to treatment.

Semen analysis. Specimens should be obtained either by masturbation or by coitus interruptus. Chemicals are present in rubber sheaths which bring the spermatozoa to a standstill and they should consequently

never be used. The specimen is collected in a glass bottle or tube warmed to blood-heat. It should then be wrapped in cotton wool, placed in a box and delivered at the laboratories within 3 hours. It is unnecessary to insist on more than 3 or 4 days of continence prior to the collecting of the specimen. Semen analysis has made such progress in recent years that it has now become a matter for experts. In this paper only a few of the main points in the examination can be dealt with.

The volume is usually between 2 and 5 c.cm. Because the accessory sexual glands are responsible for the bulk of the semen, it is generally assumed that when the volume is large the density of spermatozoa will be low. This is not necessarily the case. Dr. C. Harvey, seminologist at the Exeter Clinic, states that she has not found any relationship between density and volume, or volume and fertility, until the lower limits are reached. Undue emphasis was laid in earlier American publications on the factor of density, and the statement that unless 60,000,000 spermatozoa are found per c.cm. the husband must be considered subfertile is certainly erroneous. Conception frequently occurs with much lower counts. It must also be remembered that different samples from the same patient show great variations of density. This may partly be explained by the testes making different contributions to the semen, and it renders it difficult to estimate the results of treatment when this is being given for oligozoospermia. We have even had cases in which complete azoospermia was revealed on one occasion and on another plentiful spermatozoa were found. It is possible that some of these extreme variations may be the result of temporary occlusion of some of the ducts. There have also been cases in which husbands have been able to supply good specimens of semen by masturbation, but when post-coital tests have been carried out on their wives no spermatozoa could be found.

After the count, motility and viability of the spermatozoa have been determined, their morphology must be carefully studied, for it is the quality of the spermatozoa rather than their number which is of primary importance. If more than 25 per cent of the spermatozoa are found to be abnormal the fertility of the husband must be suspected. Abnormal forms appear in the seminal fluid as the result of imperfect spermatogenesis, arrest of spermateliosis at an early stage with premature detachment from the germinal epithelium, and of the degeneration of what was originally a normal sperm. Cells of the spermatocyte and spermatid type are often found in association with the abnormal spermatozoa. If leucocytes are also present in considerable numbers arrangements must be made for a bacteriological investigation of the semen, as well as for a more searching examination of the genital tract. Prostatic fluid should be expressed by massage and the urine cultured. Unfortunately, it is often difficult to decide whether positive bacteriological findings are the actual cause of the infertility, or whether they are the result of accidental contamination of the semen.

Testicular biopsy. The microscopic examination of a small fragment of testis yields very valuable information. It is an operation which can be carried out under a local anaesthetic, or under pentothal, and it imposes very little hardship upon the patient. Having exposed the glistening surface of the testis through a half-inch incision, it is punctured with a pointed tenotome and the button of testicular tissue which extrudes is removed with iridectomy scissors. One of the lessons which have been learned from biopsy is that wide variations are often to be found in different areas of the testis; at one point the tubules may

be found completely degenerated and at another active spermatogenesis may still be in progress. When it is necessary to differentiate between an azoospermia due to aspermatogenesis and one resulting from blockage of the ducts, testicular biopsy should always be employed rather than Huhner's older method of testicular puncture. Biopsy also allows one to decide whether the infertility is such that no treatment is likely to have effect, or whether it may be possible to increase its activity by hormone therapy.

Treatment. So little is known of the aetiology of the great majority of cases of infertility that any departure from general health, however trifling it may seem and however little related to the genital organs, must be regarded as being of importance. It would indeed be a wise step to have all subfertile patients carefully investigated by a good general physician before any special treatment is carried out. The germinal epithelium is so sensitive to toxins that all infective processes must, if possible, be eliminated. At one time I called attention to the danger of using sulphanilamides on the grounds that they had an adverse effect on spermatogenesis. But, fortunately, this seems to be of a temporary nature, and if infections cannot be got rid of by other means, they should be used. Dr. Wiesner has advocated also the use of an old-time remedy, namely, garlic, either as a fresh infusion of the herb brewed by the patient, or as an extract. I have also had cases of infected semen which have markedly benefited from the use of penicillin.

The diet, habits and mode of life of the patient are also of importance, but I have seen no case of infertility that could be attributed solely to dietetic deficiency. In spite of this I have put patients frequently on wheat germ oil capsules, more especially during the winter months. Recently it has been suggested that because the testes are very sensitive to heat the taking of prolonged hot baths should be discouraged. There can be no doubt that adverse changes are produced in the seminiferous tubules of animals by a slight increase of heat, and in spite of the fact that we have no direct evidence that hot baths are responsible for human infertility, this suggestion should not be discounted. It is also probable that the substitution of a healthy outdoor life for an indoor occupation would have a beneficial effect, but, unfortunately, it is seldom possible to arrange for this change.

Poor potency may also have to be treated. His wife's ardent desire for a child may render a nervous husband so anxious that not infrequently his potency becomes impaired. Some of the more weakly-sexed husbands seek to derive compensation for infrequent intercourse from the idea that it enriches the quality of the semen, but recent work at Cambridge and elsewhere shows that this is not the case. It has even been suggested that the high incidence of abnormal spermatozoa is more likely to occur when intercourse takes place only at long intervals.

Hormone treatment. Two types of hormones have to be considered, testosterone and anterior pituitary-like hormones. Thyroid has only a minor place in treatment, namely, in comparatively rare cases of hypopituitarism and occasionally, as an adjuvant, when the potency of a middle-aged husband is on the wane. Testosterone may have some action on the testis but its chief use is in stimulating the activity of the accessory glands. Its action on the epididymis is particularly helpful. This organ is not merely a convoluted duct which provides a convenient storage place for spermatozoa. It has its own secretions and has an important function in the bringing of spermatozoa to maturity. This being the case, poor action on its part is probably an important cause of asthenozoospermia. I

am of the opinion that an emulsion of crystalline testosterone is preferable to the usual oily solution, because it provides a deposit from which hormone is slowly absorbed for several days after an intramuscular injection. An injection of an emulsion therefore acts in the same way as the method of implanting pellets advocated by Deanesley and Parkes. The right dosage of emulsion would appear to be about 2 c.cm. weekly. Testosterone should be used in cases of low viability, poor invasive capacity, when potency is weak and when a temporary occlusion of the ducts is suspected. It is unfortunate that no satisfactory and inexpensive anterior pituitary-like hormone is yet available. The preparations of which I have most experience are Gestyl (Organon) and Synapoidin (Parke, Davis). It is very difficult to assess the true value of these preparations since they are often given in the absence of any exact knowledge of the state of the tubules. Because of this, a failure to stimulate spermatogenesis may often be due not so much to an inefficiency of the hormone as to the fact that the tubules are completely degenerated.

As in the case of the ovary, attempts are being made to stimulate the activity of the testis by carefully-regulated doses of X-rays, but more experience of this treatment must be obtained before its value can be assessed.

Surgical treatment. This plays little part in the treatment of male infertility. Since Hagner published his encouraging results interest in the operation of vasoepididymostomy, as a means of overcoming a blocked epididymis, has been renewed. It cannot be said that the results obtained in this country up to date have been satisfactory. Whilst the bringing down of a retained testicle at an early age may undoubtedly safeguard its future spermatogenic function, I have not seen any case in which this operation, when performed on an adult, has resulted in the appearance of spermatozoa in hitherto sterile semen.

Artificial insemination. This is used when the husband is suffering from incurable impotence and also in certain cases of oligozoospermia and asthenozoospermia. Should the oligozoospermia be associated with any infection of the semen, this must be eliminated or, at any rate, the infecting organism must be identified before insemination is carried out. Otherwise insemination would carry with it some risk of infection to the wife. By employing insemination it can be ensured that some spermatozoa at any rate reach the cervical canal. Before using this method every effort must, of course, have been made to improve, by treatment of the husband, the quality of the semen. But that insemination is a useful sequel to such treatment is shown by the fact that conception has occurred after it with as low a count as 2,000,000. When insemination is used for impotence the husband must not be so sexually abnormal that he is unable to produce semen by masturbation. I know of no means of obtaining semen when the husband is unable to masturbate.

The diagnosis and treatment of infertility in the male has made considerable progress during the last 5 years, but little light has yet been thrown on its causation. In many cases the genitalia appear normal, the patient is in excellent general health, he gives no history of having suffered in the past from any of the diseases that are known to have an adverse effect on fertility, and yet when the semen is examined it is found to be of very poor quality. Until more of the factors responsible for infertility are discovered, the treatment of the male, in a childless marriage, cannot be said to be satisfactory.

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Acute Renal Failure Complicating Abortion

BY

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ACUTE renal failure is rare in pregnancy. A study of the literature shows it is still rarer in abortion. The condition seems to be increasing in frequency. During the last 2½ years, 6 cases of acute renal failure complicating abortion occurred in the Gynaecological Department of the Kingston County Hospital, out of a total of 500 abortions admitted. We have also included 1 interesting case of a multipara who developed acute renal failure following the injection of sterile glycerine into the cord for delayed separation of the placenta.

The following is a synopsis of the cases:

CASE 1.

E. J., aged 19, single, primigravida, was admitted to hospital with mild pyrexia and offensive haemorrhagic vaginal discharge. Sulphapyridine, 7 g. and sulphathiazole, 12 g. were administered. Patient's blood was Group A and she was transfused with Group A blood after cross grouping. Three days later vaginal haemorrhage occurred, so digital removal of necrotic offensive placental tissue was performed under intravenous pentothal.

A vaginal smear showed numerous organisms and a culture did not grow haemolytic streptococci. Urinary tests and output were normal on admission. On the 2nd day *Bact. coli* urinary infection developed. Patient was given 19 g. of sulphonamides in all, in 3 days, with alkalis and fluid. By this time toxic signs (vomiting and dermatitis) developed and the drugs were stopped. A blood-count showed secondary anaemia with leucocytosis. Haematuria appeared on the 6th day after admission. Oliguria set in on the 7th day (30 ounces intake; 2 ounces output in 24 hours). The blood-pressure varied between 120/60 to 130/70. Vomiting became troublesome. The blood-urea rose to 320 mg. per cent. Bilateral ureteric catheterization was performed on the 8th day to exclude renal blockage by sulphathiazole.

Both kidneys were shown to be excreting blood-stained urine, which contained granular casts, leucocytes, red-blood corpuscles, *Bact. coli* in right, none in the left ureteric specimen. A 5 per cent dextrose saline intravenous drip was given. The patient's condition continued to deteriorate, so as a last measure bilateral decapsulation with left nephrostomy was performed. The kidneys were found to be congested. Two days later the urinary output increased temporarily, but coma and death occurred on the 17th day. Autopsy performed by Dr. Rushton revealed grossly enlarged oedematous kidneys with many infarcts. The uterus showed severe infection and contained small fragments of placental tissue.

Dr. Bywaters reported as follows on the histology: "The liver appears normal except for postmortem autolysis and gas bubbles due to intramortem *welchii* infection. The kidneys also show a few gas bubbles, but for the most part they are well preserved and show an appearance similar to those we have found in 'crushing injury', mismatched

transfusion, and septic abortion. There is a marked increase in interstitial oedema with some cellular infiltration, most marked in the boundary zone, and surrounding degenerative changes in the tubules in this area. Some of these tubules are necrotic. The second convoluted tubules show much desquamation; some of the cells show a brown-red pigment, others show mitotic figures of an abnormal type. The collecting tubules contain desquamated cells, partly pigmented and conglomerated masses of brown-red pigment. Some show hyperplasia of the lining and some a mass of necrotic, cellular debris and polymorphs in the lumen. (Case 1: Figs. 1 and 2.) All these appearances have been noted in crushing injury. The clinical account suggests 4 possible causes for the anuria: (1) chemotherapy; (2) mismatched blood-transfusion; (3) haemolysis due to *Cl. welchii*; (4) septic abortion kidney. There is little evidence to incriminate the first three. We would think this renal lesion was the same as we have seen in other cases of septic abortion, and that there was not any evidence of sulphonamide kidney or mismatched transfusion."

CASE 2.

H. A., aged 31, 3-para, was admitted as a 12-weeks complete septic abortion. There was a history of vaginal interference by douching with carbolic acid, sickness for 14 days, delivery of the foetus and placenta at home and bleeding for several days.

Previous history: No toxaemia, no kidney disease, malaria 7 years ago. Investigation revealed *Bact. coli* pyelonephritis with suppression of urine and uraemia. Blood urea before transfusion was 300 mg. per cent. There was a severe secondary anaemia. Her blood-pressure was 140/90.

On vaginal examination there was slight bleeding, the uterus was bulky and there was some right-sided parametritis. A blood-transfusion of 2 pints of Group O whole blood was given without reaction, followed by a 5 per cent dextrose-saline drip with high doses of vitamin B and C. The patient's condition was serious; she was drowsy, apathetic and vomiting profusely. Her optic discs showed nothing abnormal. Bleeding from the gums was present. Slight vaginal bleeding was controlled with ergometrine. The urinary output improved on the 2nd day, but the blood-urea values continued to rise for the next 4 days to 370 mg. per cent. The vomiting improved on the 3rd day and disappeared on the 4th day under therapy which included vitamin B, 40 mg., vitamin C, 1,300 mg. Her general condition rapidly improved and diuresis was normal by the 3rd day. On the 15th day after admission her blood-urea was 25 mg. per cent and her blood-pressure 120/80.

Cystoscopy and bilateral ureteric catheterization showed only cloudy urine coming from the right ureter. The sediments and cultures from the right ureter showed an inflammation of the right kidney without bacterial growth; those from the left ureter a moderate *Bact. coli* infection. An intravenous pyelography did not show any abnormality. The Wassermann and Kahn reactions were negative.

The patient was discharged in a satisfactory condition 23 days after admission. Five months later she was re-admitted for investigation of her kidney function. This revealed normal diuresis, urea concentration test values within normal limits, blood-urea 34 mg. per cent. Urine: deposit showed nothing abnormal. Urine cultures: heavy B. coli growth. Treatment with alkalis was adopted.

We concluded, therefore, from the above history and clinical findings that the patient had suppression of urine with uraemia after toxic and secondary *Bact. coli* pyelonephritis, following self-induced septic abortion in the 3rd month of pregnancy.

CASE 3.

Ch. E., aged 45, married, 11-gravida, admitted as an emergency on May 7th, 1943, with an inevitable abortion of 24 weeks. She had not had any antenatal care.

Previous history: 7 normal deliveries, 5 full-time babies, alive; 2 children died of prematurity. Recenty had noticed oedema, and some years ago had pyelitis. Three abortions, the last 2 at Kingston Hospital in 1938, 24 weeks, spontaneous, no toxaemia; 1939, 24 weeks, spontaneous, no toxaemia.

Present history: first vaginal bleeding on morning of admission associated with abdominal pain. Passed urine normally at home, had no swelling of the ankles or face and felt well.

General condition on admission: Pale and and moderately shocked. Optic discs normal, blood-pressure 120/70; temperature 97.4°F.; urine, trace albumin. Fundus, 26 weeks, uterus tender.

Vaginal examination, slight loss; cervix, 1 finger dilated, uterus contracting.

Conservative treatment was adopted. Blood-transfusion by drip, 2 pints Group O blood, not cross-matched, no reaction.

The following day, May 8th, a foetus, freshly dead, 24 weeks, was expelled with an incomplete placenta. The placenta showed widely spread, fresh, red infarcts and a large recent clot covering one third of the organ. In view of free loss, dilatation and curettage were performed under 0.5 g. pentothal intravenous anaesthesia. Remnants of placenta were removed and the haemorrhage was controlled. Three pints Group O blood were transfused by drip without incident. Sulphathiazole, 1 g., t.d.s. was given for prophylactic reasons with sodium citrate, gr. 30, t.d.s. Patient was fairly well and passed urine. On May 9th, oliguria was noticed in the evening. The patient's condition seemed satisfactory. Sulphathiazole was stopped after a total dose of 4 g. On May 10th, a sudden eclamptic convulsion occurred, lasting 3 minutes. Later in the day catheterization revealed anuria. Patient's general condition was now poor, and her blood-pressure had risen to 200/120. Blood examination showed severe secondary anaemia with leucocytosis and a blood-urea of 124 mg. per cent. Further serological investigations by Dr. Loutit showed that the transfused blood was Group Rh positive and the patient's blood was Group Rh positive also. The serum did not contain any atypical agglutinins. The Van den Bergh blood-bilirubin test was direct and indirect negative. This negative reaction within 36 hours after the last blood-transfusion showed that there was little likelihood of an appreciable haemolysis of the transfused blood. Treatment consisted of 40 c.cm., 30 per cent intravenous dextrose and by drip in the form of a 5 per cent solution.

Both ureters were catheterized, but only a few c.cm. of blood-stained urine escaped. A diagnosis of symmetrical cortical necrosis of the kidneys was therefore made.

As the suppression of urine with signs of uraemia did not improve, Mr. Jennings Marshall performed a bilateral decapsulation with right-sided nephrostomy under local anaesthesia. Both kidneys appeared congested and the capsule stripped easily. There were multiple small haemorrhages over the surface. The patient died 12 hours later in uraemic coma.

Postmortem examination (Dr. Rushton) disclosed a left-sided pleurisy and left ventricular hypertrophy with mild atheroma of the aorta and oedema of the brain. The left and right kidneys showed extensive subcapsular haemorrhages with irregular wedge-shaped multiple necrotic areas in the cortex. The liver was markedly degenerated. The uterus did not show any abnormality. Dr. Loutit, who submitted the sections to Dr. W. Woods of the Pathological Department of the London Hospital, and Professor Ellis of the University of Oxford, sent us the following reports on the specimen from the autopsy:

"One portion of the kidney: Large wedge-shaped, dark purplish-red area of subcapsular haemorrhage. Irregular, more or less wedge-shaped, multiple patches in cortex, separated by dark areas showing radial striation and separated from the medulla by similar striated dark zone. Vessels not unduly prominent. Several other portions of the kidney showing similar picture. Portion of arterial wall: Showing faint, linear streaks in intima and oedematous adventitia. Portion of the liver: Well-marked lobular pattern, the periphery of the lobules being putty colour and the centres pale brown. Portion of left ventricle: Showing pale, firm musculature. Kidneys: The histological picture in the sections of the two kidneys is similar. The capsule is replaced by a layer of recent blood-clot. The cortex is divided by bands of haemorrhage into roughly wedge-shaped areas which show almost complete coagulative necrosis (Figs. 3 and 4) the ghosts of glomeruli and tubules being well visualized, but individual cells are not recognizable and nuclei, where visible, are very pale, swollen and structureless or fragmented and pyknotic. In the haemorrhagic areas the parenchyma is also necrotic, the interstitial tissue being markedly infiltrated with polymorphonuclear leucocytes, lymphocytes and mononuclear cells as well as red-blood cells. The tubules of the medulla show degenerative changes, karyolysis being marked; they contain hyaline, granular and occasional cellular, and blood-casts (Fig. 5). There are also a few small interstitial medullary haemorrhages. In the intermediate zone is a narrow band of cortical tissue in which the cells of the tubules are not completely necrosed though the nuclei are markedly pale. The

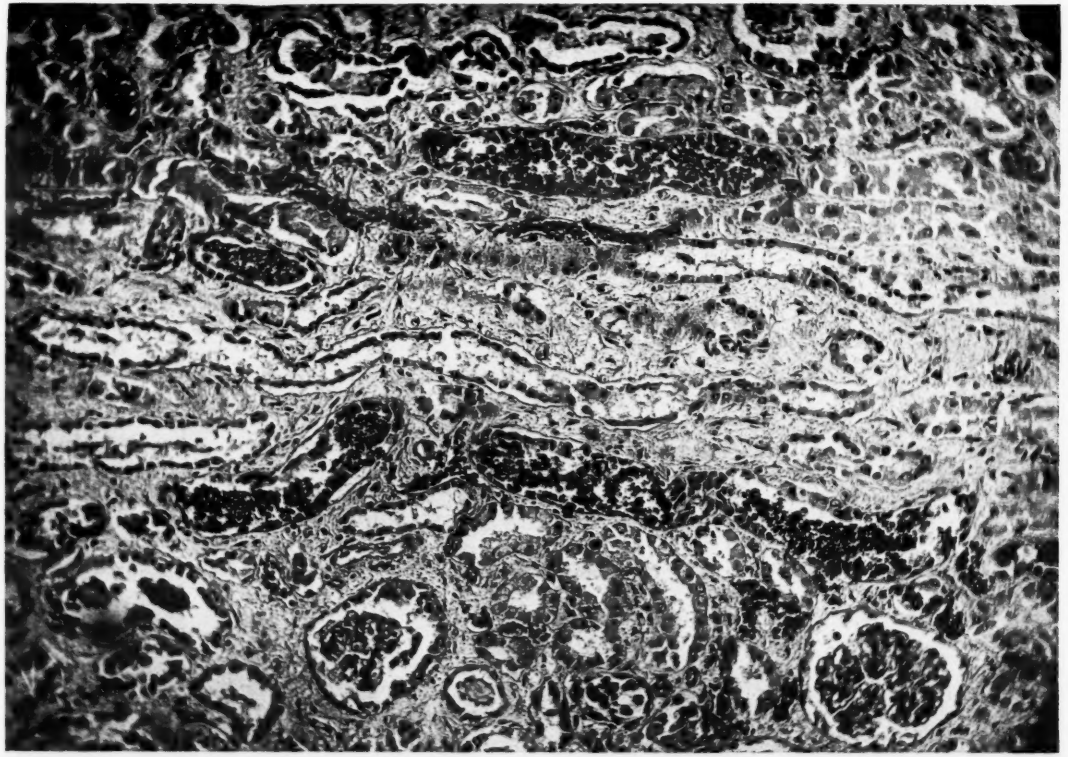


FIG. 1.

Photomicrograph of a kidney section in Case 1 (abortion kidney) showing interstitial oedema, cellular infiltration and degenerative changes in the tubules. (Magnification × 160)

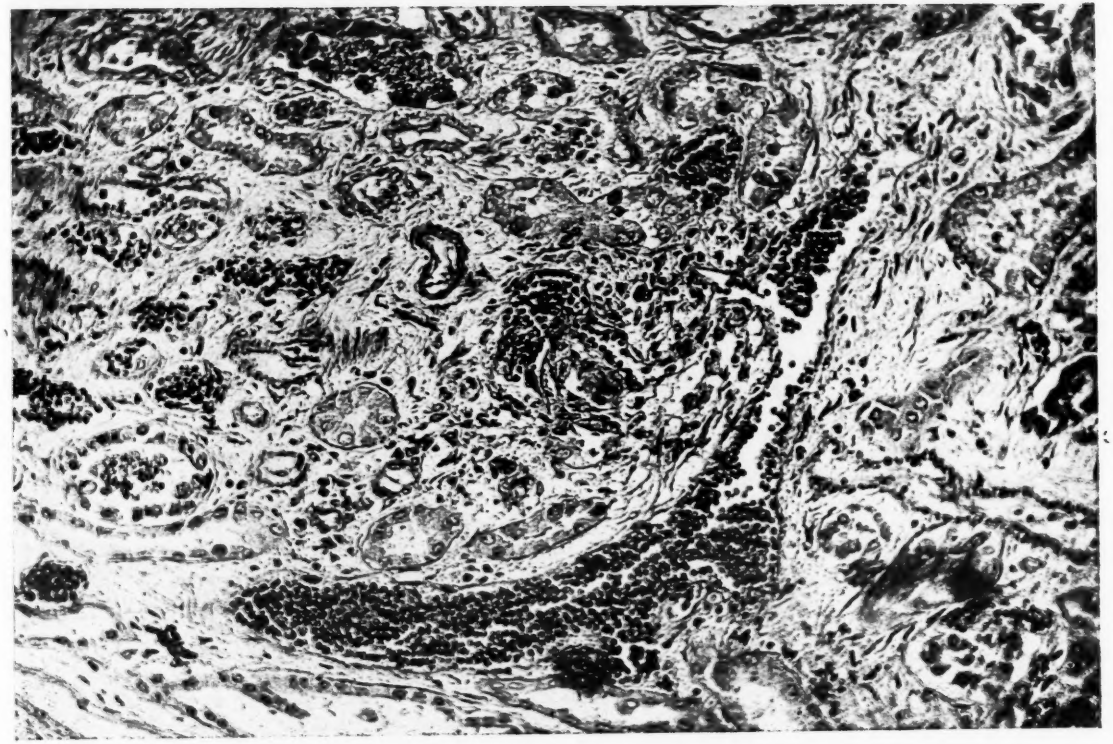


FIG. 2.

Photomicrograph of an " abortion kidney " showing high power magnification (× 220) of an area with pigmented ribbon and granular casts.

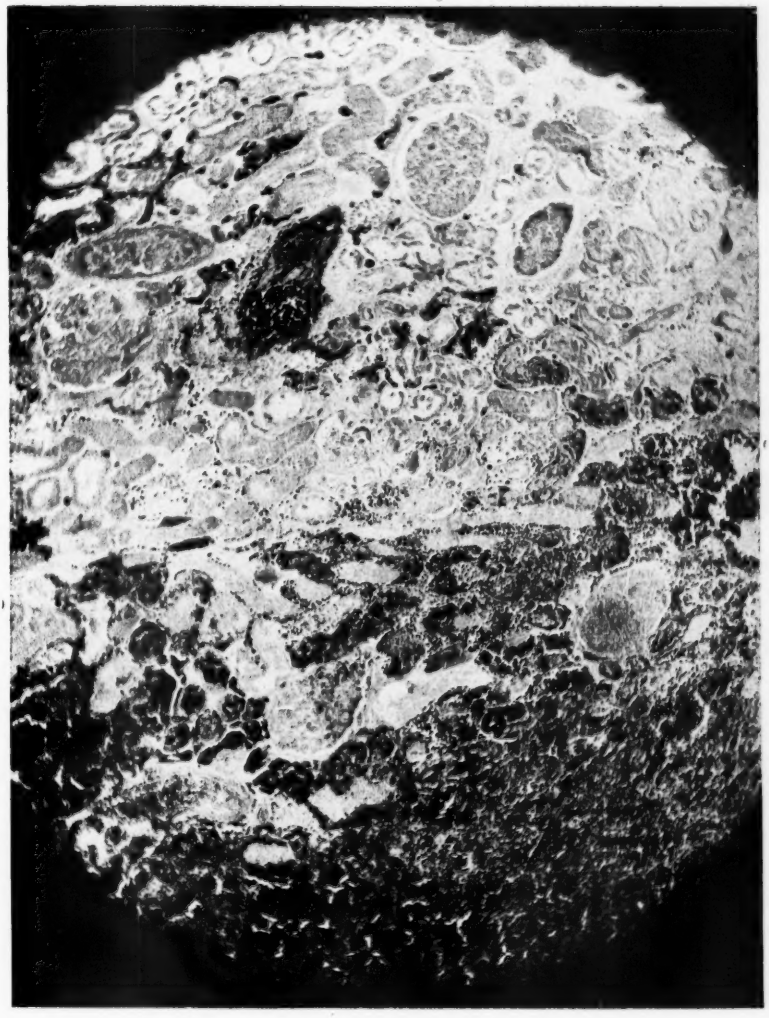


FIG. 3.

Photomicrograph of a kidney section in Case 3, showing almost complete coagulative necrosis. (Magnification × 65)

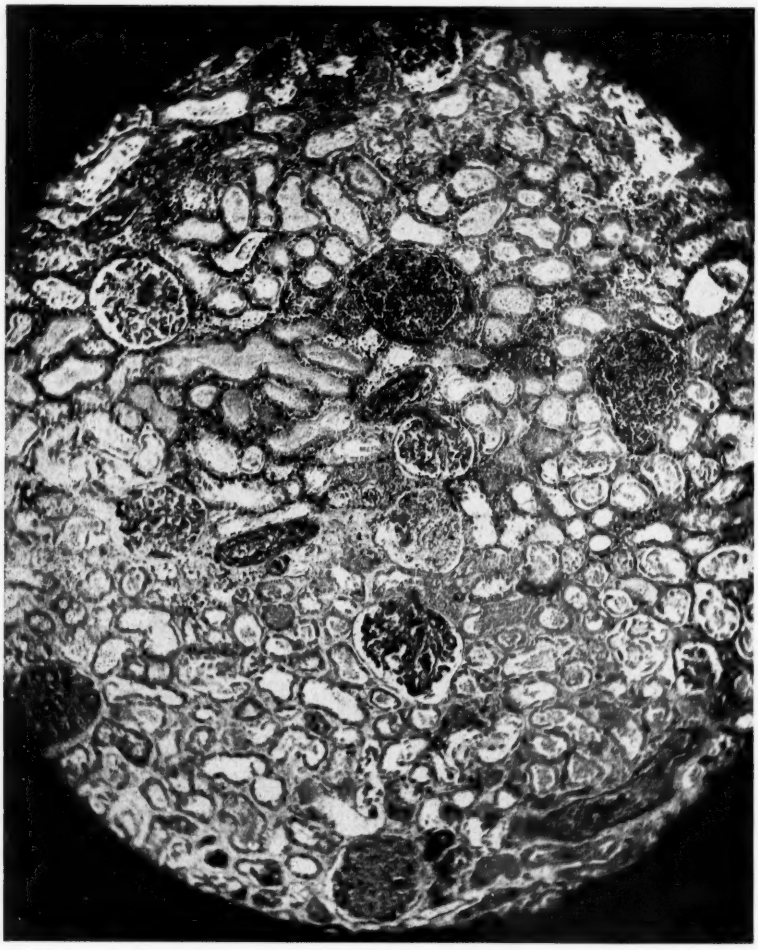


FIG. 4.

Photomicrograph of a kidney section in Case 3, showing the ghosts of glomeruli and tubules. (Magnification × 60)

FIG. 5.

Photomicrograph from section of kidney in Case 3, showing the degenerative changes of the tubules and thrombosis of an interlobular artery.

(Magnification × 75)

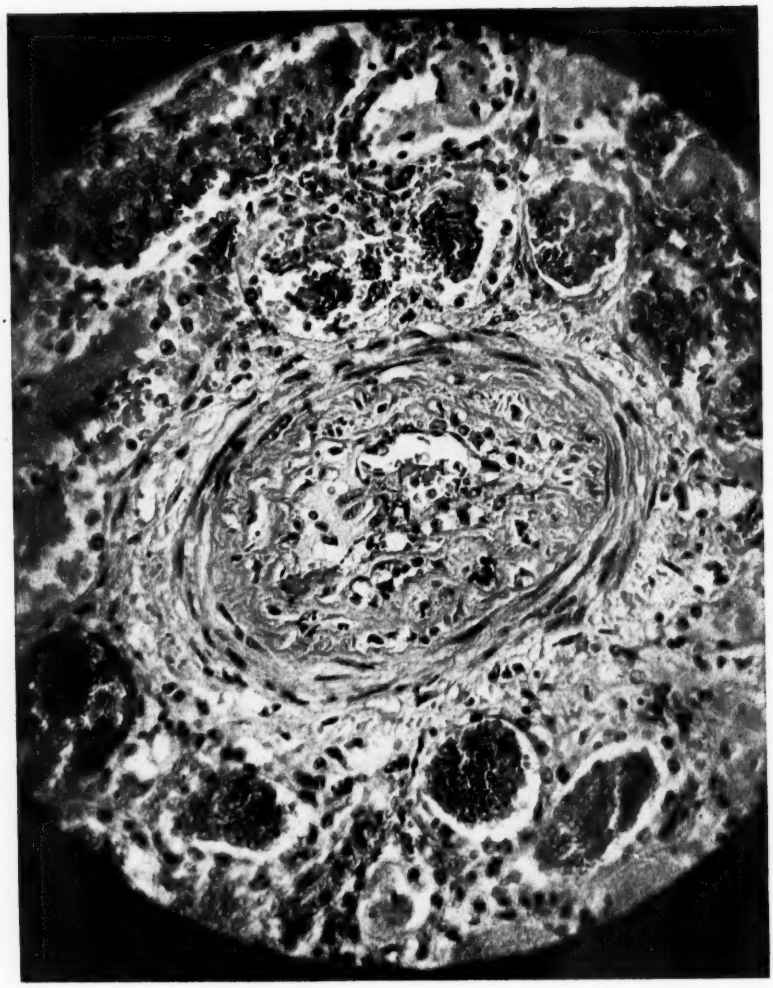


FIG. 6.

Photomicrograph of a kidney section in Case 3 showing intravascular thrombosis. (Magnification × 270)

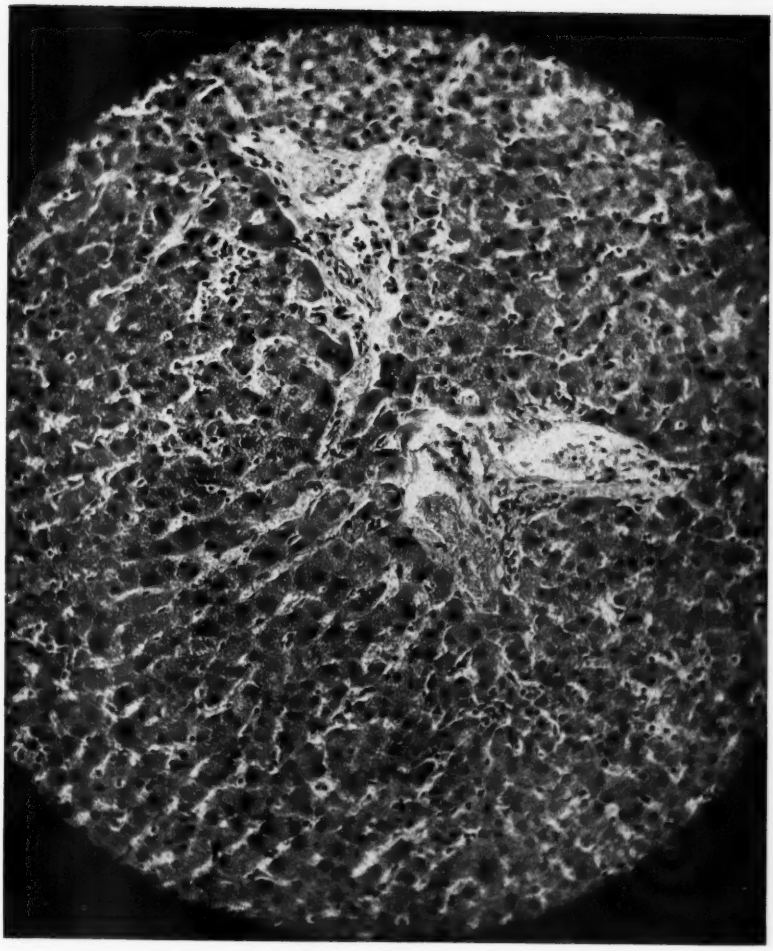
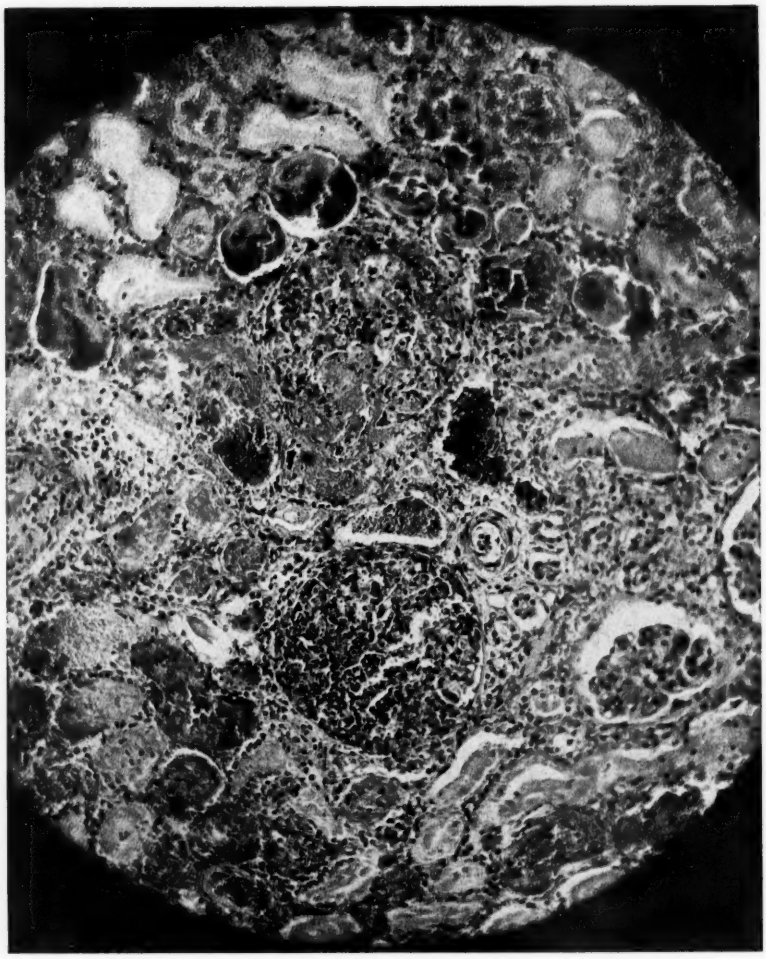


FIG. 7.

Photomicrograph of a liver section in Case 3. (Magnification × 130)



CASE 3, FIG. 4.

Photomicrograph of a kidney section in Case 3 of bilateral cortical necrosis in abortion. (Magnification × 130)

glomeruli here show focal necroses, adhesions to Bowman's capsule and polymorphonuclear infiltration. Some show acute degeneration and necrosis of the afferent arterioles, the almost structureless swollen arteriolar wall staining positively for fibrin. The interlobular arteries of the cortex show similar fibrinoid degeneration of the intima and necrosis of the whole arterial wall; there is no perivascular cuffing. Intravascular thrombi are seen in many arteries (Fig. 6). Thrombi are also seen in some of the arcuate arteries of the intermediate zone, but apart from some splitting of the internal elastic laminae the coats of these vessels appear normal. Liver: A section of the liver (Fig. 7) shows the cells of the parenchyma to be markedly degenerate, with palely staining, granular and faintly vacuolar cytoplasm. Sudanophil fat is not demonstrable. The nuclei are markedly swollen and show loss of chromatin. The cells appear to be more or less equally affected throughout the lobules. The cells in the centres of lobules contain a fairly large amount of fine, yellowish-brown pigment. In many of the portal canals the portal vein is seen to be filled with a fine reticulum taking a positive fibrin stain and enmeshing a variable number of red cells. Heart: The myocardial fibres appear of average size, a few of the nuclei are pale and vesicular. A large branch of the coronary artery appears normal."

From a review of the clinical features of this case it is evident that the patient, an elderly multipara, had some preceding renal involvement and an abortion taint in her history.

Case 4.

J. E., aged 31, married, 2-para; was admitted after 14 weeks amenorrhoea as a septic incomplete abortion.

On admission the patient was anaemic and shocked, with blood-pressure 110/70; pulse-rate 120, pyrexia 100.6°F., haemoglobin 32 per cent, micturition normal, and the uterus enlarged to the size of a 14 weeks' pregnancy. She gave a history of having used slippery elm on herself to bring on abortion. Swabs and smears from the vagina showed no haemolytic streptococci.

An immediate blood-transfusion of 2 pints Group O whole blood was given by the drip method.

Sulphapyridine, 1 g. 4-hourly with sodium citrate, gr. 30 t.d.s. and pituitrin, 3 units b.d. were given. The treatment was conservative for 14 days in view of the obvious signs of uterine infection. Three further blood-transfusions, by drip, with 2 pints of Group O whole blood after cross-grouping (Group O) were given to treat the marked secondary anaemia. Only after the 4th blood-transfusion (2 pints, Group O cells) did the patient react with headaches, right loin pain and slight jaundice. On the 15th day after her admission dilatation and curettage was performed after the administration of 0.4 g. sodium pentothal. The sulphonamides had been discontinued several days before, after a total dose of 26 g.

On the 17th day after admission, 3 days after her last transfusion, the patient showed almost complete suppression of urine and severe jaundice. Her blood-urea at this stage was 205 mg. per cent. Serological investigations by Dr. Loutit proved that the urinary suppression in this patient was due to the transfusion of Rh incompatible blood. The fact that the recipient was Rh negative and that her serum 3 days after the transfusion contained an antibody which agglutinated both random Rh positive erythrocytes and erythrocytes transfused 3 days previously, suggests that the Rh antibody developed as a result of the immune process from the 3 previous transfusions and was the antibody responsible for the haemolytic reactions. A bilateral splanchnic block, with 1:2000 Amethocaine, was performed and a 5 per cent dextrose-saline drip was set up. The patient responded very satisfactorily. Her diuresis improved remarkably (5 ounces after the block) and her severe anaemia was treated with a further blood-transfusion of 2 pints Group O Rh negative cells and whole blood, without incident. The further course of the patient was uneventful and she made a perfect recovery. She was discharged 8 weeks after admission and had a blood-urea of 25 mg. per cent. The patient did not attend for follow-up examination.

Case 5.

S. E., aged 37, married, 3-gravida; admitted as an emergency without previous antenatal care on October 4th, 1943, complaining of moderate vaginal loss.

Previous history: Three normal deliveries; the last was in 1940, twins and toxaemia; the first was in 1930. No abortions. She had no history of any

disease. One month before the patient fell and sustained a superficial wound on her left leg which healed. At that time she had a slight show which disappeared on resting.

Present pregnancy: For 1 month she had noticed occasional swelling of her legs, also headaches, which were relieved by aspirin. On the day of admission a sudden heavy loss occurred, accompanied by abdominal pains. Foetal movements were felt until the day before admission. On general examination the patient was pale, but her condition was satisfactory, her blood-pressure 110/70, temperature 101.2°F., pulse-rate 93; heart, lungs, and central nervous system were normal. Her uterus was enlarged to about the size of a 24 weeks' pregnancy, and was contracting. No foetal heart could be heard. On vaginal examination the cervix admitted 1 finger.

A diagnosis of septic inevitable abortion was made.

On catheterization: Three ounces of deeply blood-stained urine were withdrawn. A vaginal swab and smear were negative. Sodium citrate, gr. 30, 4-hourly and sulphathiazole, 1 g., t.d.s.; pitocin 3 units, b.d., Benerva 5 mg. intramuscularly were ordered and a self-retaining catheter was inserted.

On October 5th the patient was vomiting and complained of abdominal pains due to uterine contractions. There was also moderate bleeding. Blood-pressure 110/70, temperature 100.8°F., pulse-rate 110. Only 3 ounces of deeply blood-stained urine were collected from the bladder in 24 hours. Urine analysis showed blood pigments and debris only. Culture revealed no growth. Patient's blood-urea was 25 mg. per cent and her haemoglobin (Haldane) 68 per cent. Sulphathiazole was discontinued after a total dose of 4 g. 40 c.cm. of 30 per cent dextrose was given intravenously, followed by 5 per cent dextrose-saline by intravenous drip. At 10 p.m. the patient delivered herself of a freshly macerated foetus of about 24 weeks, with a normal-looking placenta and a few old clots. The total blood-loss was small and the uterus contracted well after 5 units of pitocin. A slight rigor and vomiting occurred after the delivery. October 6th: Severe oliguria (2½ ounces) continued throughout the day. The urine was dark red in colour. The blood-urea was 72 mg. per cent. Copious vomiting persisted. October 7th: Condition of patient deteriorating. Vomiting still constant and severe; drowsiness developing. Urinary output dropped to 5½ drachms in 24 hours. Tests showed that the patient was Group O Rh negative. An X-ray of the kidneys revealed nothing abnormal. To effect diuresis a bilateral splanchnic block with 70 c.cm. ½ per cent procaine was performed. October 8th: Suppression of urine (1 ounce) continued. On spectroscopic examination the dark urine showed oxyhaemoglobin but no methaemoglobin. Patient complained of drowsiness, headaches and spots before the eyes. She vomited 24 ounces during the day. Optic discs: no papilloedema. A complete blood-count showed haemoglobin 60 per cent, 3,740,000 red cells, 10,800 leucocytes. A cystoscopy and bilateral ureteric catheterization were performed and it was found that the right ureter passed only very little blood-stained urine, the left nothing. October 9th: Oedema of the legs appeared. Patient's condition was uraemic. Suppression of the urine continued; only 1 ounce in 24 hours was passed. October 10th: 3.5 ounces of blood-stained urine was passed, blood-urea 75 mg. per cent. October 12th: Patient seemed moribund with respiration laboured and profuse vomiting; she was irrational and drowsy; her blood-pressure was 130/70. The urinary output was 2.4 ounces and on analysis showed many pus cells and red cells, no casts, a few renal epithelial cells, no growth, no tubercle bacilli. Another bilateral splanchnic block was performed; this time with 60 c.cm. of 1:2000 Anethaine (Amethocaine) Glaxo. October 13th: Less vomiting (15 ounces), passed 6.4 ounces of blood-stained urine. October 14th: Marked improvement in general condition. Vomited only 3 ounces. Passed 17½ ounces of clear urine. Blood-pressure 130/70. Optic discs normal. October 15th: Normal mental condition, no uraemic symptoms. Passed 36 ounces of urine during the day. October 16th: Patient felt very well. Blood-pressure 130/70; blood-urea 180 mg. per cent. Urinary output increased to 75 ounces of cloudy urine. Analysis showed pus and red cells and heavy growth of B. coli. Although her haemoglobin was only 60 per cent, a blood-transfusion was not given in view of the Rh negative test, to avoid possible reaction in the damaged kidneys. During the following days her condition improved steadily. The treatment consisted of large doses of iron, vitamins C and B_1. Her Wassermann and Kahn reactions were negative.

It is an interesting fact that from November 5th the patient developed hypertension. The blood-pressure rose from 130/96 to 160/90; the urine was clear, the output was within normal limits and the blood-urea was 30 mg. per cent. She was discharged on November 27th in excellent health, after being in hospital for 56 days. We were unable to see her for follow-up examinations, but from a recent letter we assume that she is getting on well.

This is one of the rare cases of "symmetrical cortical necrosis" of the kidneys with recovery.

CASE 6.

K. L., aged 36, primigravida, admitted August 8th, 1944, with septic incomplete abortion of 20 weeks. There was nothing of note in her previous history.

Present history: Patient stated that 10 days ago rupture of the membranes occurred. She did not interfere with herself and did not see any foetus come away. She had passed urine normally.

On admission: General condition was satisfactory; urine normal; temperature 100°F.; pulse-rate 106; blood-pressure 100/70; uterus the size of 20 weeks; few small fibroids also palpable in fundus. Sulphapyridine, 1 g. 4-hourly with alkalis and abundant fluids were ordered. August 9th: Digital removal of the placenta and curettage was performed, accompanied by free bleeding which was controlled by intrauterine injection of pitocin, ergometrine and a hot proflavine douche. A catheter specimen showed nothing on culture. A vaginal swab and smear was negative; blood-transfusion was avoided until Rh factor was determined. On the next day pyrexia 101.6°F.; pulse-rate 120; general examination discovered nothing abnormal. August 11th: The lower abdomen was tender and patient complained of a sore throat. Micturition was normal and painless. On August 12th she vomited twice and felt ill. Sulphapyridine was now replaced by the less toxic Sulphamezathine. On the 13th, oliguria developed for the first time, 12 ounces in 24 hours. The urine was not blood-stained. Sulphonamides were discontinued after a total dose of 21 g. After this diuresis improved speedily, but her vomiting continued on the 14th. Oedema of the legs became apparent and slight right papill-oedema was found. Her blood-pressure was 96/40. A catheter specimen did not reveal any abnormality. Her blood-urea was 42 mg. per cent and her blood Group O Rh positive. The full blood-count revealed a moderate secondary anaemia and leucocytosis, and it was therefore unlikely that the renal failure was due to the sulphonamides. She was treated by intravenous 5 per cent dextrose-saline and alkaline therapy and she made a speedy recovery.

The patient's condition was of interest in so far as it probably indicated mild, early kidney damage due to a septic abortion. One month later a general examination did not reveal anything abnormal and an intravenous pyelography was normal.

CASE 7.

N. T., aged 28, married, 2-para. After normal antenatal period admitted on April 24th, 1935, in labour to the City of London Maternity Hospital. A normal delivery of a 6 pounds 7 ounces girl occurred but the placenta was retained. Four hours after the end of the 2nd stage 200 c.cm. sterile glycerine were injected into the umbilical cord without effect. The patient complained of headache and had a slight loss of blood *per vaginam* following the injection. April 26th: Placenta still retained. Patient vomited once and was jaundiced. Temperature 98.8°F., pulse-rate 92. A glucose-saline infusion was given. On manual removal of the placenta a contraction ring and a small interstitial fibroid were found. At the end of the operation the patient was very shocked; pulse-rate 130, skin cold and clammy. She recovered with warmth, etc. April 27th: Vomiting marked; oliguria now present; urine port-wine coloured (1½ ounces), which on microscopy showed clumps of red cells. Potassium citrate given. April 28th: Complete suppression of urine developed. Treatment consisted of saline and sodium bicarbonate transfusion. One rigor occurred. April 29th: Blood-urea 120 mg. per cent. Blood-sugar 0.1 per cent. Van den Bergh: direct negative, indirect positive. Haemoglobin, 35 per cent; red cells, 1,900,000; white cells, 13,700; polymorphs, 85 per cent; lymphocytes, 13 per cent, mononuclears, 2 per cent. Catheter specimen of urine: no pus, blood or casts, some diphteroids only. Later catheter specimens: pus, few red cells, no casts, no leucin or tyrocin or bile, pH 8. Mixtures of organisms, methaemoglobin present. Passed 1 to 2 ounces urine into bed. Blood-pressure 100/65. Bowels opened. April 30th: Blood-urea

134 mg. per cent, no vomiting, less jaundiced. There was a faint trace of albumin. The patient was drinking well. Her abdomen was distended but not tender. May 1st: Oedema of the face appeared, anuria and uraemic smell were also present; the bowels were loosely open. Intravenous saline with dextrose administered. The patient passed 3 ounces of urine, blood-urea 127 mg. per cent. May 2nd: Haemoglobin, 40 per cent; red cells, 2,500,000; colour index 0.8; anuria, bowels open. May 3rd: Rigor, oedema of the legs and face; right lung congested, cardiac stimulants, glucose 30 per cent, 50 c.cm. given; blood-pressure 140/75; blood-urea 241 mg. per cent; haemoglobin 38 per cent; patient developing acute mania—morphine and hyoscine were ordered. There was still suppression of urine; plenty of fluids taken by mouth. May 4th: Definite improvement, wet bed twice. Chest still congested and no movement of the bowels; blood-pressure 150/85, caffeine given. May 5th: Passed urine 3 times (8 ounces) and one stool, rigor, temperature 100.2°F., pulse-rate 140, irrational. May 6th: Patient very ill—gasping. May 7th: Some improvement noted. General oedema now present, no jaundice, cyanosis marked. Patient was given cardiac stimulants, magnesium sulphate, calomel. Output of urine was 25 ounces. May 8th: Urine: specific gravity 1012, pH 8.5, cloud of albumin, no acetone. Blood-urea 298 mg. per cent. Excellent urine output, cough improved, no headaches, clean tongue, oedema nearly gone. May 9th: Urine 60 ounces, blood-pressure 135/70. Abdomen still enlarged, ascites present. Uterus involuting slowly, haemoglobin 52 per cent, white cells 16,000, red cells 3,050,000. May 10th: 130 ounces urine in 24 hours. Excellent condition. May 11th: 127 ounces urine. Blood urea 270 mg. per cent. May 12th: 110 ounces of urine. May 13th: 120 ounces of urine. Blood-urea 20 mg. per cent. May 15th: Urine specific gravity 1014, pH 5. No sugar, no acetone, cloud of albumin. May 16th: Haemoglobin 54 per cent, urine output well maintained. May 20th: Catheter specimen: heavy growth of B. coli, no blood, no casts. May 21st: blood-urea 60 mg. per cent, patient on iron and potassium citrate. May 31st: Blood-urea 28 mg. per cent. June 3rd: Blood-pressure 115/75. June 7th: Haemoglobin 68 per cent, red cells 3,700,000; white cells 9,000. June 12th: The patient was discharged.

Postnatal examination. September 24th, 1935: Patient was very well, blood-urea 28 mg. per cent, blood-pressure 135/70, urine clear, optic discs: faint blurring, micturition normal, no anaemia, menstrual periods 3-5/31. June 21st, 1936: Blood-pressure 128/80; urine clear; occasional headaches; otherwise quite well.

This case of acute suppression occurred after delivery at term in a healthy multipara. Apparently the origin of the trouble was the sterile glycerine given for the retention of the placenta. Clinically she was a case of bilateral cortical necrosis. A study of the available literature has failed to reveal a similar case.

Review of the Literature on Renal Failure Following Abortion.

Symmetrical cortical necrosis of the kidneys was first reported by Juhel-Renoy[1] in 1886 in France, and by Bradford and Lawrence[2] in 1898 in England. Torrens[3] described a 29-year-old patient with a 4 months' miscarriage followed by anuria. There was mild pyrexia but no convulsions. Postmortem examination disclosed a massive infarction of the entire cortex of both kidneys and thrombosis of the renal and ovarian vessels. Jardine and Kennedy[4] reported a 23-year-old primigravida who for several weeks before admission had had oedema, vomiting, failure of vision and headaches. She aborted at 4½ months, when her urine was solid with albumin. She then had one eclamptic fit followed by 2 days anuria. On postmortem examination the liver changes were not typical of eclampsia. The kidneys showed patchy cortical necrosis. Thrombosis was very infrequent. White's[5] cases of puerperal anuria with recovery were not regarded by Gibberd[30] as typical of symmetrical cortical necrosis according to the published biopsy which was taken after decapsulation. Cruickshank[6] in 1923 described a 10-gravida of 33 with a 5 months' abortion who died after 4 days' oliguria and 5 days'

anuria. Postmortem examination showed symmetrical cortical necrosis of the kidneys and widespread thrombosis of the smaller renal arteries.

Fraenkel[7] encountered haemoglobin casts in postabortal gas-gangrene septicaemia and blamed the low pressure due to the illness for the diminished secretion and the haemoglobin casts. Carson and Rockwood[8] reported a case of a 36-year-old woman with a hypertension of 200/130 and anuria in an abortion of 24 weeks. No other signs of toxaemia, no convulsions, no abruptio placentae were present. A symmetrical cortical necrosis of the kidneys was found on postmortem examination. Seitz[9] reported 3 cases in which quinine, given in 3 doses of 0.5 g. (gr. $7\frac{1}{2}$) to induce abortion in early pregnancy, was followed by suppression of urine with fatal haemoglobinuria and methaemoglobinuria. No histologic changes were described. Kutz and Traugott[10] reported the same picture. Scriver and Oertel[11] examined carefully 2 abortions with anuria. Both cases, 34 and 33 years old, aborted with accidental haemorrhage at 24 weeks without any signs of toxaemia apart from hypertension. There were no fits. The kidneys in both cases were typical of cortical necrosis. Petri[12] published a case in early pregnancy with oliguria, haemoglobinuria and methaemoglobinuria after barbital and 1 g. quinine each day for 3 days. Microscopically the kidneys showed abundant haemoglobin casts in the glomeruli and in the convoluted tubules. The tubules did not show any epithelial changes. The paper by Kellar and Arnott[13] in 1932 deserves special reference. Their case (1) was a 26-year-old 1-para, whose pregnancy in the 5th month, with moderate signs of toxaemia and suppression of urine for a fortnight at home, was terminated by an abdominal hysterotomy. Death in coma occurred after 18 days anuria. Their case (3) was aged 29, 2-para, pre-eclampsia, blood-pressure 158/100, urine solid with albumin, oedema and oliguria. After concealed accidental haemorrhage, pregnancy was terminated at 5 months. Almost complete anuria followed for 7 days. Ureteric catheterization and decapsulation failed to relieve the suppression of urine and the patient died. Both cases showed evidence of toxaemia; the second, premature separation of the placenta. Pathological examination of the kidneys showed classical cortical necrosis with a predominating selective vascular element. As the probable mechanism of thrombosis they assume an "endotheliolytic toxin" (Leith Murray) manufactured in the liver or placenta. Warner and Hibbitts[14] in 1932 reported a multipara, 26 years old, with a 6 months' miscarriage and typical signs of abruptio placentae and severe pre-eclampsia; blood-pressure 180/110; albuminuria, oliguria, oedema, non-protein nitrogen 55 mg. per cent. Spontaneous abortion of a dead foetus occurred with typical infarcted placenta and large blood clot. After 3 days of complete suppression of urine, death in uraemia supervened. In their case, also, the renal cortical changes showed distribution on a circulatory basis. They consider that vasoparesis with stasis and thrombosis are the aetiological factors of necrosis. Jeddeloh[15] published the first article on renal pathology after septic abortion, following 2 cases of criminal attempts at termination of pregnancy in the 4th and 3rd months. In the first case the uterus and gut were perforated. Eight days after hysterectomy the patient died of anuria. The postmortem examination showed cortical necrosis of the kidneys with severe inflammatory reaction. Case 2 died of anuria 4 days after an attempted criminal abortion completed by dilatation and curettage. Again a symmetrical renal cortical necrosis was found and Jeddeloh observed inflammatory

changes in the glomeruli, first convoluted tubules and interstitial tissue. Jeddeloh cited the only previously recorded cases of this renal condition from the German literature: Hertzog,[16] who described bilateral necrosis of the renal cortex following a purulent peritonitis in a girl of 10 years, and Furtwängler[17] after a severe accident. Lubarsch,[18] and Fahr,[19] described similar pictures in toxaemia of pregnancy.

Bowes[20] described a 6 months' miscarriage in a patient of 32 years, with complete anuria, toxaemia and no convulsions or antepartum haemorrhage. Johnstone[21] thought that the loss of blood leads to slowing of circulation and thrombosis, otherwise it would be difficult to understand the selective action of "endothelial" toxins in cortical necrosis. Ash[22] again referred to the prolonged vasospasm as the cause for the occlusion and called symmetrical cortical necrosis "angioneurotic anuria."

In a clinico-pathological study in 1934 one of us (W.S.)[23] reported haemoglobinuria and methaemoglobinuria with suppression of urine in a patient 7 months pregnant with toxaemia of pregnancy. The urine was burgundy-coloured and contained many haemoglobin casts, granular casts and red cells. A quinine intoxication was diagnosed and the patient died in coma without convulsions. The postmortem examination showed typical eclamptic liver changes, a cerebral haemorrhage and severe degeneration of the kidney tubules in the absence of blockage. In 3.572 g. weight of different organs, 0.3 g. (5 gr.) of quinine could be recovered and it was suggested that in this case of "eclampsia without convulsions" quinine taken by the patient caused a paroxysmal destruction of erythrocytes.

In the light of more recent research on the mechanism of the production of the renal lesion in haemolytic conditions, the case is both rare and interesting. It demonstrates severe toxic tubular damage without mechanical blockage of the tubular lumina by the altered blood pigments. The primary mechanism of the renal lesion was probably the toxaemia with its haemolytic and necrosing factors aggravated by the haemolytic action of quinine causing massive haemoglobinuria and methaemoglobinuria. The findings seem to support the view that simple mechanical blockage of the tubules by blood casts is not responsible for the anuria in fatal cases of haemoglobinuria in pregnancy. The exact nature of quinine haemolysis is not understood.

Nocht and Kikuth[24] have shown that very small quantities of quinine may facilitate amboceptor haemolysis in animal experiments. Donath and Landsteiner[25] found that during pregnancy autohaemolysins are formed by a gradual disintegration of a large number of erythrocytes. The only report in the American literature of fatal haemoglobinuric kidney damage due to quinine in an abortion comes from Terplan and Javert.[26] Their case was a 41-year-old woman in the 3rd month of pregnancy who had taken quinine, estimated as 100 gr. (6.5 g.). She developed vaginal bleeding, oliguria, haemoglobinuria, and died in uraemic coma. Microscopically the kidneys showed marked distension of the tubular system brought about by masses of haemoglobin casts associated with definite focal inflammatory lesions; 3 gr. (0.2 g.) of quinine were recovered from the liver. The authors stressed the fact of special susceptibility to quinine haemolysis in pregnancy. Terplan and Javert commenting further on the renal failure, state that quinine does not exert a direct action on the kidneys and is excreted by the gastro-intestinal tract and the kidneys very shortly after intake. When the haemoglobin, following haemolysis, reaches a sufficient level in the blood it is excreted by the kidneys and haemoglobinuria results. They regard the

kidney damage as due to the accumulation of haemoglobin in the tubules, with mechanical obstruction of the outflow of secreted urine.

Reyna[27] published a typical case of pre-eclampsia and placenta praevia in a 24 weeks' miscarriage. The blood pressure was 245/180. Death in coma followed 3 days' anuria. The renal pathology showed symmetrical cortical necrosis and the liver showed eclamptic changes. Kernau[28] reported 4 cases of gas-gangrene after abortion with oliguria and anuria. All cases showed severe nephrosis and haemoglobinuria with casts. It is interesting here to note that Kernau cites a publication by Wolff[29] who published, from the war of 1914–18, 100 cases of wound gas-gangrene, in 38 of which the author found similar kidney changes.

Gibberd[30] who reported 2 cases of symmetrical cortical necrosis in late pregnancy with recovery, pointed out the importance of making a clinical diagnosis of symmetrical cortical necrosis and not abandoning it in the fortunate event of the patient's recovery, for that of "suppression of urine". He remarked on the pathognomonical initial secretion of a small amount of bloodstained urine in the presence of the other typical clinical features in cases of concealed accidental haemorrhage.

The cases of Crook,[31] Scriver and Oertel,[11] Gibberd,[30] Dieckmann,[32] Madding, Binger and Hunt,[33] Dingle,[34] Adam[35] our cases 5 and 7 are, so far as we could detect in the available published literature, the only clinically diagnosed cases of symmetrical cortical necrosis in pregnancy with recovery. Volk's[36] case is an abortion of 6 months with premature separation of the placenta, toxaemia and anuria of 5 days duration. Rubin and Petru's[37] case was another 24 weeks abortion with anuria and no signs of accidental haemorrhage and toxaemia.

In 1941 Duff and Moore,[38] pathologists at McGill University, Montreal, gave an excellent review of the whole subject of symmetrical cortical necrosis of the kidneys, including the previous survey by Ash in 1933. They analysed 71 cases of bilateral cortical necrosis and divided them into 2 groups:

(1) Forty-eight cases occurring in pregnancy.

(2) Twenty-three cases occurring apart from pregnancy.

Two-thirds of the pregnancy series terminated in abortion or premature labour. In 21 cases there was antepartum haemorrhage and in 7 cases, pre-eclampsia.

Only 1 out of the 12 authors, in the 15 cases of cortical necrosis in abortion, mentions convulsions in the clinical features. Jardine and Kennedy's case of a 4½ months abortion with toxaemia and anuria had one eclamptic fit and the post-mortem examination did not show typical eclamptic changes in the liver.

In Duff and Moore's 15 collected cases of cortical necrosis, the abortion was associated:

in 4 cases with toxaemia;
in 6 cases with accidental haemorrhage;
in 3 cases with infection;
in 2 cases with no manifest clinical conditions.

The most puzzling feature of symmetrical cortical necrosis in abortion is its rarity compared with the frequency of its apparent exciting causes, such as retroplacental haemorrhage, shock and infection. Duff and Moore described the pathology of the kidneys very clearly. These authors agree as to the localisation of the vascular changes in the intralobular arteries and efferent arterioles of the glomeruli and they also agree to the virtual exclusion of arteries of larger size. The aetiology of the condition is still in doubt although it is generally agreed that symmetrical cortical necrosis

is the result of ischaemia caused by obstruction of the terminal cortical arteries and arterioles. The usual stages seem to be vasospasm, vasoparalysis, stasis, thrombosis and necrosis.

Dawbarn and Williams[39] noticed severe oliguria with passing of black urine in gas-gangrene infection of the uterus.

In some cases in which the Cl. welchii infection had been successfully controlled, Adey[40] considered that anuria caused the death of the patients. He pointed out that the kidney changes were due to the direct action of the toxin and to the disintegration of haemoglobin in the tubules.

In the recent excellent review on Cl. welchii gas-gangrene infection of the uterus by Rendle-Short[41] we searched the 4 reported postabortal cases for evidence of renal failure.

In the 3 Bristol cases, Case 1 procured an abortion by injecting cow's urine into the uterus. The patient showed only a little bloodstained urine on catheterization and the postmortem examination of the kidneys was not mentioned.

Case 3 (1942) when $3\frac{1}{2}$ to 4 months pregnant, syringed with soap and water and later passed acid, port-wine-coloured urine which was not noticeably reduced in quantity. The deposit showed blood pigment, debris, bloodcasts and Cl. welchii in large numbers.

Rendle-Short discussed the urine in a typical case of gas-gangrene but we are unable to find notes of any renal investigations or on renal pathology elucidating the renal failure in postabortal gas-gangrene cases. In future it would be of value to have full reports on diuresis and renal investigations in this particular field of renal failure.

A very interesting contribution to the problem of postabortive anuria came from a group of South American investigators. Chabanier, Lobo Onell, Michon and Lelu[42] gave a very thorough study of the decapsulated kidneys in a case of criminal abortion. The patient's main symptoms were, apart from haematuria and anuria, an accentuated hypochloraemia due to persistent vomiting. They considered that the latter caused the renal insufficiency. A tubular nephritis, interstitial oedema with moderate infiltration of inflammatory cells and very few glomerular lesions were found. Normal blood-chlorides were established by hypertonic saline infusions and the patient recovered quickly.

Renal insufficiency may be one of the outstanding complications of haemolytic reactions, after transfusion of incompatible blood in pregnancy. De Gowin and Harden[43] reported in a study of 2,128 transfusions of stored blood that haemoglobinuria occurred in 5 (0.23 per cent), and death in 2 (0.09 per cent) cases. Berstein[44] and Brainard[45] reported anuria in pregnancy following transfusion of compatible blood as determined by our present agglutination tests. Wiener and Peters[46] reviewing intragroup haemolytic reactions after Landsteiner and Wiener's[47] discovery of the presence of an iso-glutinin, anti-Rh, classify them into (1) those occurring in patients receiving repeated transfusions of Rh positive blood and (2) those occurring in patients in or after labour who have not received previous transfusions. Our case 4 is a classical example of the mechanism responsible for the renal failure, an "immunisation of the Rh negative patient to the repeated transfusion of the Rh positive agglutinogen". That such intragroup haemolytic transfusion reactions may occur especially in Rh negative mothers whose Rh positive foetuses were affected with erythroblastosis or haemolytic anaemia of the new-born was supported by Levine,[48] Levine, Burnham, Katzin and Vogel,[48a] Wiener,[49] Boorman, Dodd and Mollison,[50] Loutit,[51] Harrison and Meacock,[52] and others.

The occurrence of isohaemolysins, alpha and beta, in higher titres in postpartum women may provide an explanation for the specific stimulation in hetero-specific pregnancy: Group A or Group B foetus in a Group O mother. (Wiener and Peters,[46] Jonsson.[53])

The subject of renal failure after incompatible blood transfusions was further reviewed by Jewesbury,[54] Wiener and his co-workers, De Gowin,[55] Kilduffe and De Bakey,[56] so that we indicated in our paper only the features of particular interest to the obstetrician.

Butler, Danforth and Scudder[57] recently presented a case and a review of the Rh factor in intragroup blood transfusion reactions in obstetrical patients. The treatment was directed towards correcting the acidosis, maintaining fluid and electrolyte balance, and restoring blood volume and plasma proteins. Adrenal cortical extract and hypertonic saline were administered to cause sodium retention and potassium diuresis, to protect the patients from the convulsive action of the increase of potassium salts in the blood, a method worth remembering. Among 25 similar cases selected from the literature, haemoglobinuria was noticed in 12, convulsions in 10, hypertension and oedema in 6. Rapid progression to suppression of urine was present in 3 fatal cases. There were 5 deaths in these 25 cases.

Reference seems necessary to the present conflicting views on the mechanism of production of the renal lesion and the suppression of urine by an incompatible transfusion in pregnancy.

Two explanations are given:

(1) Mechanical blockage of the tubules by acid haemoglobin. (Ponfik,[58] Baker and Dodds,[59] De Gowin and co-workers, de Navasquez.[60])

(2) Irritating or toxic products released by the haemolytic reaction have a direct effect on the kidneys (Bordley,[61] De Gowin and co-workers, Hesse and Filatow[62]).

The renal lesion is characterized by oedema and cellular infiltration of the interstitial tissues, degeneration and necrotic changes in the collecting tubules, deposition of pigmented casts, dilatation of the convoluted tubules. The glomeruli are normal. The pathology of the kidneys resembles strikingly the pathological findings in "crush injury".

Godwin and McCall[63] have described cortical necrosis following perforated gastric ulcer with peritonitis and made bacterial toxins responsible for the glomerular capillary dilatation. They stressed that shock with the resulting capillary stasis and haemoconcentration may have been a contributary cause of renal ischaemia.

Bratton[64] described anuria with casts not associated with blood transfusion in 9 fatal cases of antepartum bleeding. The case records showed that 4 patients had "septic abortion" with suppression of urine and one had raised blood-urea. Postmortem findings were tubular degeneration, necrosis of occasional cells, formation of cellular and haematogenous casts, especially in the collecting and discharging tubules, unaffected glomeruli, interstitial oedema with small foci of subacute inflammatory infiltration in relation to the blocked tubules. Apart from engorgement the vessels showed no other abnormality. Bratton's microscopical description differs from the previous pathological findings of Jeddeloh.[15] Bratton pointed out that the histology of the kidneys suggests a toxic lesion, but as the onset of anuria may precede abortion the toxic process is not necessarily puerperal in origin but may be initiated previously.

We think that one cannot rule out the question of drug-ingestion in some of Bratton's cases of septic abortion, and this

factor, therefore, may be responsible, too, for the postmortem picture.

The third type of suppression of urine in early pregnancy, obstruction to the outflow of secreted urine, is a subject that has given rise to much discussion and stimulated many investigations since 1939.

In 1941 Bywaters and Beall,[65, 65a] and their associates at the British Postgraduate Medical School, published a paper showing the close association of crush injuries with renal failure. After the heavy air-raids on London the attention of the medical world was focused on that problem.

Bywaters *et al.*,[66] Dunn *et al.*,[67] Bywaters and Dible,[68] Darmaddy *et al.*,[69] and several others gave a comprehensive description of the pathology of the kidney damage, consisting of degenerative and necrotic tubular lesions localized most frequently in the ascending limbs of Henle and the second convoluted tubule, with brown casts (myohaemoglobin) in some tubules.

Young and McMichael[70] stressed the analogy of the shock-azotaemic state following the trauma of difficult labour with the crush syndrome.

Young[71] referred to the similarity of biochemical and renal changes in the "crush syndrome" and the syndrome in concealed accidental haemorrhage. In his papers Young[72] has given us much enlightenment on the causes of renal failure after uteroplacental damage. He drew the following conclusions:

Concealed accidental retroplacental haemorrhage is a massive utero-placental lesion of ischaemic origin in which the acute renal lesion with haematuria, oliguria, suppression and uraemia may ultimately develop. The toxic substance giving rise to tubular degeneration is derived from tissue autolysis. Shock and haemolysis, so often present, may have a similar origin.

In an analysis of the aetiology of renal failure in 79 cases of accidental haemorrhage in late pregnancy, 5 cases (6.5 per cent) showed renal impairment. Among 59 consecutive cases of obstetrical haemorrhage (postpartum, placenta praevia, accidental) in only 5 of the 10 accidental was renal impairment demonstrable. Young concluded, therefore, that shock from haemorrhage has no essential part in the initiation of the renal lesion. The second factor, haemolysis with tubular blockage, is also not solely responsible for the renal failure following accidental haemorrhage. Young postulated a third factor: a toxic material is generated in the dying retroplacental concealed haemorrhagic uteroplacental lesion which is responsible for the renal syndrome in accidental haemorrhage. The analogy is drawn from the "nephrotoxic theory" in crush syndrome where the source of the toxin has been regarded as ischaemic muscle.

The latest description of 2 cases of renal failure following abortion comes from Dingle.[34] One case, a 6 months pregnancy, had a severe pre-eclamptic toxaemia, in which the added trauma of induction produced shock and kidney failure. The postmortem examination disclosed complete bilateral cortical necrosis. The second case was that of a patient who fell, and probably had a traumatic accidental haemorrhage, which produced haematuria, shock and oliguria with uraemia. The patient made a complete recovery after 10 days oliguria and uraemia (blood-urea up to 356 mg. per cent). In both cases there was haematuria, followed by oliguria and a marked rise in blood-urea following delivery, which was accompanied by a sudden alarming drop in blood-pressure due to shock.

In the second case there was a marked tendency to thrombosis with lowering of the blood coagulation time, a complication noted also by Davidson and Turner[73] and Gibberd.[30]

Dingle indicates, from a study of the

recent literature, that the aetiology of the condition may lie in a damaged placenta. Her cases seem to offer convincing evidence for those who regard renal anoxia secondary to hypotension (shock) as the mechanism of renal failure.

Although cortical necrosis of the kidneys could be produced experimentally by *Staphylococcus aureus* toxins (Von Glahn and Weld,[74] De Navasquez[60a]), surprisingly none of the cases described in the last 10 years were associated with septic abortion, a fact that seems worth mentioning, especially since Dunn and Montgomery[75] reviewed in their 8 cases of renal cortical necrosis a variety of infections, such as sore throat, scarlatina, post-operative streptococcal infection, pneumonia, diphtheria, "grippe" and dysentery.

It is interesting to note here that sulphapyridine anuria has a similar mechanism and clinico-pathological picture (Ravid and Chesner[76]), and in cases of septic abortion under treatment with sulphonamides it is essential to exclude the sulphonamides as a possible factor in the urinary upset.

Darmaddy, *et al.*,[69] have recently (1944) reported that they have seen many cases with extensive muscle ischaemia and necrosis without traumatic uraemia.

The "toxic substance" has not yet been identified, so the theory is still *sub judice*.

In the last 2 years we have failed to find signs of renal failure in many cases of difficult labour with severe trauma (failed forceps, severe lacerations, etc.).

Reference is made to a rare cause of oliguria in early and late pregnancy—allergic or angio-neurotic oedema—by Seley.[77] The existence of oliguria and anuria due to toxaemia in early pregnancy is rare. Acute glomerulo-nephritis, pyelonephritis, dehydration, hyperemesis gravidarum, etc., are conditions which it is not our intention to discuss in this paper.

Treatment.

The salient features of the conservative treatment of oliguria and anuria following abortion consist in:

(1) Ample fluid intake to overcome the negative fluid balance. Dieckmann and Kramer[78] defined oliguria as the excretion of less than 600 c.cm. of urine within 24 hours, and anuria as the absence of urine for 12 or more hours. We have found that 5 per cent hypertonic dextrose in physiological saline in amounts of 3 to 4 pints daily by slow intravenous drip has been the most efficient means of increasing diuresis and replenishing the mineral content of the plasma.

(2) Alkaline fluids by mouth, prophylactically, for recipients of blood-transfusion, in the form of sodium citrate 90 to 180 gr. daily. Two per cent sodium sulphate intravenously can be given as a stimulating diuretic. We have had no experience with lactate solutions which were strongly recommended by Dieckmann and Kramer.

(3) High doses of ascorbic acid as a diuretic, and vitamin B_1 to prevent or improve severe vomiting. Both proved useful in the reported cases.

From the operative measures which have been advocated and reported as successful in the treatment of anuria, decapsulation, splanchnic block and cystoscopy with ureteral catheterization were employed in our cases.

(4) All cases of septic abortion or of shock were put on intake and output charts and had the blood-urea and catheter specimens of urine examined as a routine.

Decapsulation was attempted in Cases 1 and 4 of our series. We could not convince ourselves that the kidneys in these cases were under tension at operation and we considered the operation tilted the scale rapidly against the patients. Gibberd[30] has already given very strong evidence from the

pathology of the fallacy of employing decapsulation in cortical necrosis. There is nowhere in the literature the slightest proof that mechanical compression from without cuts off the blood-supply to the glomeruli. The vessels were the very opposite of compressed; they were engorged. The clinical course, biochemistry and histology of cortical necrosis, points to glomerular failure as the cause of progressive renal insufficiency, so that mechanical pressure on the tubules by the acute swelling of the confined kidneys is quite unconvincing as a cause of anuria.

Cabot and Iber,[79] who presented the largest single collection of cases of total anuria from different causes at the Mayo Clinic, do not believe that decapsulation has any place in the modern treatment of this condition.

Madding and his associates[33] advise conduct of the treatment of anuria in pregnancy on modern conservative lines, as in their own successful cases.

Kellar and Arnott[13] had discouraging results with decapsulation in cortical necrosis and advised staving off anuria by other means.

Johnstone[21] was impressed with isotonic sulphate solution as a means of stimulating diuresis in a probable case of postpartum cortical necrosis.

Among the 48 cases of symmetrical cortical necrosis in the pregnancy of Duff and Moore's series, Crook's case is the only one which recovered after decapsulation. Of their 15 fatal cases of renal cortical necrosis following abortion, there was not a single one in which decapsulation was of avail.

A further analysis of the reported 8 recoveries of clinically diagnosed cases of cortical necrosis in pregnancy showed that only Crook's patient had a decapsulation—another argument against a surgical procedure which should have no place in the treatment of renal failure in pregnancy.

Splanchnic Block. The limitations of existing methods of treatment of oliguria and anuria in pregnancy encouraged us to attempt, in two of our most serious cases, a bilateral posterior splanchnic block, according to the method of Kappis. James[80] had recently described excellently the technique of this type of regional analgesia.

Our case 4 was a septic incomplete abortion with severe haemolytic transfusion reaction, due to Rh incompatibility, in which bilateral splanchnic block with 1:2000 Anethaine Glaxo (an analgesic solution of amethocaine hydrochloride, with the addition of adrenaline in 1:200,000 concentration), was performed to stave off renal failure. The response was rapid and the patient recovered. Case 5 was a patient with septic inevitable abortion and symmetrical cortical necrosis, who responded dramatically to a second bilateral splanchnic block with 1:2000 Anethaine Glaxo, after all conservative measures and a first splanchnic block with ½ per cent procaine had failed to promote diuresis. The literature contains only a few reports of splanchnic nerve block as a method of treatment of suppression of urine.

Neuwirth[81] published the first conclusive evidence of the relief of reflex-anuria by means of splanchnic block in anuria. Havlicek,[82] Rubritius,[83] and Haslinger[84] were other continental writers who confirmed the usefulness of splanchnic block in anuria.

Peters[85] presented a case of prolonged anuria after blood transfusion, in which a single splanchnic block, after failure of other measures, produced diuresis within a few hours. He explained the mechanism of splanchnic block as the breaking of the reflex arc through which sympathetic reflexes pass. Experiments show that irritation of the splanchnic nerve causes oliguria, and section of the nerves conversely, polyuria. (See Bernard,[86] Milliker and Karr,[87] and Caldwell, Marx and Rown-

tree.[88]) Peters also discussed the advantages of splanchnic nerve block over spinal anaesthesia, which was recommended by Hayes and Paramore,[89] and Cubitt.[90]

Russian authors (Hesse and Filatow[62]), made experimental observations that renal vasospasm occurs in haemolytic reactions as a result of the liberation of depressor substances acting directly on the vessel walls, with consequent impairment of kidney function. They were impressed with the results of immediate transfusion of compatible blood in small quantities experimentally and clinically. Among 16 cases of severe renal failure following blood transfusion, they had only 2 deaths. Visneoskji[91] treated cases by splanchnic block successfully.

To be efficient, spinal block must be at a high level because some fibres making up the splanchnic nerve arise from as high as the 4th dorsal segment. Splanchnic block gives a more complete interruption than spinal anaesthesia which works as high as the 6th dorsal segment only.

In our experience splanchnic block does not produce motor weakness or paraesthesias. Both our patients were able to move about in bed and there was no appreciable fall in the blood-pressure.

Definite conclusions cannot be drawn as to the exact mechanism of splanchnic nerve block in the relief of suppression of urine in pregnancy, due to cortical necrosis. The predominant vascular element—ischaemia—is striking and arteriolar and arterial spasm may be the first events to determine all the later cortical lesions. The block, should, therefore, be used early.

The renal picture in dogs and rats in the classical Goldblatt[92] experiments, after clamping the renal arteries, is not unlike that in our described cases of cortical necrosis. Splanchnic block may be "releasing the clamp" which produces renal anaemia. We know Goldblatt's experiments are stated not to be due to nervous phenomena. Splanchnic block is not a panacea for all cases of oliguria or anuria, but, to our knowledge, these are the first 2 cases of suppression of urine in early pregnancy in which conservative methods and splanchnic nerve block have given good results and probably prevented a fatal result.

Catheterization of the ureters was employed in 4 of our cases, prior to any other surgical procedure, first to assist in the diagnosis of the renal pathology, secondly to exclude blockage of the ureters. In both cases of cortical necrosis the operation appeared to fail. In the first case of "septic abortion kidney" with blockage in the convoluted tubules by masses of pigment, it was also impossible to promote diuresis by catheterization of both ureters.

Discussion and Summary.

A clinical, and in part, pathological study of the 7 cases we have collected shows that renal failure is one of the most serious of the complications of pregnancy. Although the condition has been well known for many years, the clinical picture has gained more interest since the publication of papers dealing with traumatic uraemia ("crush syndrome") in this war.

Age. The ages of the patients ranged from 19 to 45 years. The majority of the patients fall in the higher age-groups.

Gravida. There were 2 primiparae in the group. All the other patients were multiparae. Thus it would appear that multiparity predisposes to these renal complications.

Stage of Pregnancy. We have found our abortion cases distributed from 12 weeks to 24 weeks. It is interesting that all cases of renal cortical necrosis occurred at a time when the placenta had already developed.

Aetiology. The cases discussed allow us to group the renal complications into those which occur in:

(1) non-septic abortions;

(2) septic abortions.

It should be emphasized that 5 out of our 6 cases of abortion were septic, a figure that indicates the importance of the infection factor in the causation of the acute renal insufficiency in this series. We cannot of course, completely exclude the possibility of the sulphonamides having some part in the aetiology.

Conclusion.

An extensive summary and analysis of the world's literature on renal failure complicating abortion has not been reported previously. Up to 1941 only 15 cases of abortion with renal failure among 48 cases occurring in pregnancy could be discovered. Since then only 2 cases (Dingle's) and our 2 have been reported. The poor prognosis is evident from the fact that up to the present time only 9 cases of cortical necrosis in pregnancy with recovery can be traced in the available publications.

An outline of the treatment of oliguria and anuria following abortion is given. Decapsulation should have no place in the treatment of renal failure in these cases. Among the 9 recovered cases of renal cortical necrosis in pregnancy in the literature, 8 recovered without this surgical procedure. Both our patients upon whom decapsulation was performed died. Two of our cases with suppression of urine and azotaemia responded excellently to bilateral splanchnic block with 1:2000 anethaine.

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The Mother in Industry *

BY

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IN the short time at our disposal I shall try to suggest subjects for discussion rather than give a detailed account of my experience as a Medical Inspector of Factories, of the mother in industry. I propose to do surface anatomy on this mother and to divide her into (a) the expectant mother, (b) the nursing mother, and (c) the mother with young children, with a foreword on the prospective mother for this is the keynote to protective factory legislation for women.

The Factories Act of 1937, the previous Factories Acts and the Orders and Regulations made thereunder had in mind the fundamental physiological function of women, that of childbearing, and to prevent any detriment to the function has determined the law with regard to women's employment generally, and specifically in those "dangerous trades" where women are allowed. Some restriction on women's employment in industry is founded on tradition, and on the supposed greater susceptibility of women to certain toxic substances, i.e. lead compounds and benzene, but in my view this is a complicated issue.

It would be difficult to prove on physiological grounds that women (apart from pregnancy) are more susceptible than men to injury where there is a health risk common to both. There are, however, in addition to general statutory restrictions on the employment of women in industry, certain special restrictions relating to particular processes to which reference must be made. These are, with one exception, lead processes, and these restrictions on employment of women have been "imposed on the ground that the use of lead involves special dangers to women."[1] If I may express my view of this official statement it would be to the effect that since lead has been used as an abortefacient, the prohibition from employment in lead processes, involving special risk, of women of child bearing age could be supported on medical grounds.

Both women and young persons (i.e. those under 18), may, however, be employed legally on work involving the use of lead compounds (approximately, material containing less than 5 per cent of soluble lead compound). Where such work is undertaken certain precautions have to be taken to control the risks from lead dust or fumes and periodic medical examination is required. Cases of lead poisoning or lead absorption among women and young persons so employed are in fact practically unknown.

Other health risks in industry are on the whole common to men and women, the factor determining ill effects being the intensity and duration of exposure to the hazard. The element of susceptibility can rarely be predetermined and is probably not influenced by sex, although it may be by habits of individuals. It has been suggested that women are more susceptible

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than men to benzene poisoning but this is not supported by a study of groups of workers of both sexes exposed to similar concentrations.[2]

It is possible, however, that women exposed to benzene may show the harmful effects in an obvious way peculiar to women —by menstrual changes. The alleged susceptibility of women to lead poisoning, as shown by abortion, may be capable of a similar explanation.

(a) *The Expectant Mother.*

There is not, contrary to the popular view, legislation forbidding the employment in industry of women during any stage of pregnancy and women, in larger numbers in wartime than in peace, have in fact been so employed. This employment is terminated either on the instigation of the worker or the employer and fortunately there is practically no disagreement between these two as to when during pregnancy industrial occupation should cease. In wartime there has been the Essential Works Order, controlling release from work, but permission to leave employment on account of pregnancy would not be withheld if desired by the woman. If she elects to remain at work against the wishes of the employer the question falls to be decided by the National Service Officer, but I think such cases are rare.

We very rarely, too, hear of accidents in industry to women who are pregnant. You might think this is odd as women are every year involved in quite a large number of industrial accidents and it might so happen that some of these women were pregnant. I think the explanation lies in the care—without the spur of legislation—that is given to women in industry in this condition, and perhaps to the fact that there is no hard and fast rule as to how long such employment may continue—each case being considered on its merits in relation to the individual's state of health, her home circumstances and the nature and conditions of her employment. These desiderata are most conveniently and efficiently satisfied when there is medical (including nursing) supervision in the factory, but such is the public conscience in this country towards the expectant mother that the consideration accorded her by the industrial laity does much to prevent injury to her, although this consideration (prompted by principles which can be well understood) may sometimes have the effect of terminating prematurely her period of useful employment.

The usual procedure where there is a medical service in the factory—including a whole- or part-time doctor and one or more nurses—is for the woman to seek advice from this department as soon as the fact of pregnancy is established. Then she is advised in general terms and as to attendance at the local authority antenatal clinic, while her work in the factory is reviewed in the light of the new circumstances. She is, or should be, removed from contact with toxic substances such as trinitrotoluene, benzene or lead compounds, and from cold, wet work, and from heavy work, particularly where this involves muscular strain of an intermittent or jerky nature, and from work which—and this is very rare in my experience if thought is given to the remedy —requires continual standing. The ideal is, of course, in a "sedentary" job to alternate sitting with standing, and we all know that exercise in moderation is good for the pregnant woman. The hours of work would, in the ideal case I am considering, be modified if necessary to the particular needs of the individual and as a rule night-employment (permitted to women during the war) would be generally avoided. You may say, and cogently, that such changes, perhaps carrying with them anything from a

diminished wage-packet to termination of employment will tend to postpone the revelation of pregnancy until it becomes apparent. This is the very thing that is to be avoided, if only on account of the relative frequency of miscarriage during the first 3 months of pregnancy. Some firms have therefore declared in advance their policy of non-dismissal (where dismissal is legal) on the ground of pregnancy, and sometimes monetary benefits are given to make up for shortened hours and change of employment. One firm, with the object of getting the women to return after childbirth, introduced a scheme by which each woman on reporting her pregnancy became entitled to £1 a week for 3 months. Most women were advised to leave this firm's employment at about the 6th month.

In general, it is the economic urge which keeps women at work after the 3rd month of pregnancy, but in this connexion it must be remembered that factory work is not necessarily more exacting than domestic work and where in a factory there is a medical service, the needs of the pregnant woman may receive more constant, expert consideration (in conjunction with the outside health service) than is possible at home.

It is not scientifically sound to determine in general terms the period of healthy employment of women in industry who are pregnant; this must, as I have suggested, be determined to suit the individual needs. We have records of women who have, without untoward incident stayed at work until the 8th month, but this is very unusual. Somewhere about the 20th to 28th week is much more common while, of course, large numbers, including those who are well provided for, leave industry much earlier.

There have been a few instances during wartime of antenatal clinics in factories. These have not set out to compete with Local Authority clinics, but the object has been to keep the workers at work and to save their time. A firm estimated that 5,244 hours were so saved in 8 months by 115 prospective mothers. The general view, however, is that medical and nursing services in factories will function best if regarded as ancillary to hospital and local authority clinics (and private and panel doctors) and that continuity of ante- and postnatal care can only thus be ensured.

What of the results of this employment? I have no statistics to give you because we are not, of course, officially informed of pregnancy in industry, and it is only by a scrutiny of reported accidents and of notifiable industrial diseases that the complicating fact of pregnancy can be revealed. My recollection over 20 years is of only a handful of such cases. One stands out in my memory—that of a well-built girl in the twenties who had been passed in the pre-war period as medically fit for her job in a sugar refining factory. At the end of the 2nd year of the war, without further medical examination, she had been put on what was described as a "man's job"—filling sugar bags and trucking these (weighing 1 or 2 hundredweights) to a chute. One day after taking a bag on to her truck, she stepped back on to the empty truck of another girl; she fell—but did not report to the ambulance room. The same evening she had a severe haemorrhage followed by a miscarriage. She was 2½ months pregnant and had not reported to the medical department of the factory.

I am telling you of one of the apparent failures of the present system, and that these are generally connected with the early months of pregnancy before the condition has been disclosed to a responsible person in industry will not surprise you. In another case (also wartime) where this also held true, the coroner's verdict was one of accidental death from cerebral oedema following abortion due to being struck at work. In this regrettable case, the woman was aged

40 and a widow—pregnancy had not been disclosed. She was working with 3 other women stacking flour bags, weighing 140 pounds, when the stack collapsed. She and another woman were struck by falling bags, the widow went home and was admitted to hospital 14 days later. Death occurred after 3 days and only at a post-mortem examination, was the fact of pregnancy (4th to 5th month) established. There had evidently been haemorrhage and a miscarriage at home.

To turn from these isolated instances to the record of one factory during wartime, there were in 6 months, among 4,905 female munition workers, 3 cases of abortion treated at the works' surgery and the same number of cases of amenorrhoea. In other words, to be forewarned of pregnancy in a factory is to be forearmed.

(b) *The Nursing Mother.*

In industry the law has stepped in and reads (Public Health Act 1936, S. 205) "if the occupier of a factory or workshop . . . . (this was before the Factories Act of 1937, after which the distinction is obsolete) . . . knowingly allows a woman to be employed therein within 4 weeks after she has given birth to a child, he shall be liable . . . etc. In fact it seems almost unknown for a woman to return to work earlier than the law allows and again, it seems that few women return, at any rate to the factory where they were formerly employed, within 6 months to a year of confinement. One factory indeed gave a maternity benefit of £6 to women who returned to work after 8 weeks. It has been estimated among one group of factories that 12 per cent only of married women returned to work after confinement, whereas the percentage of the unmarried was 20 per cent.

A few factories provided facilities in wartime for mothers to nurse their infants, but I think you will agree that the baby in the first months of life wants more than lacteal attention from the mother, and that it is not really in the best interests of either mother or child for nursing mothers to be employed in factories.

Crèches at or near factories, mostly with a minimal age for admittance of 9 months, are another matter. With individual attention under the supervision of a fully trained nurse the infant is sure of his necessities of life—food, sleep and cleanliness—and he is saved the irregularities which call down his displeasure were he at hand while mother earned the wage packet to supplement, or satisfy, the needs of the weekly budget.

(c) *Mothers With Young Children.*

These can similarly make use of nursery schools until the child is of school age. These are so much more in the province of the local authority that I will conclude by a very brief consideration of the lot of the woman in industry with young children.

Her lot is unenviable, and how she performs the double, or is it triple (with a husband at home), function, I really do not know. What is evident in industry is that the married woman has 65 per cent more absence from sickness than her unmarried sister and this is particularly noticeable in married women under 25 years old. The proportion discharged for reasons of ill health is also much higher among married women.

We shall, I suggest, never change the position with regard to sex differences in absence through sickness (the men's rate is lower than the women's) so long as women have to work away from home and yet continue to fulfil their household obligations. This position creates worse hardships for the women themselves than for the children for whom there is alternative care at home

or in nursery or national schools with the all essential food, rest and recreation.

"The longer you can look back the further you can look forward," said Winston Churchill to an audience of doctors, and it is indeed encouraging after the experiences of 2 great wars to be able to record the useful work, under medical supervision, that the expectant mother has been able to give to industry without detriment to her health.

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A Case of Aplastic Anaemia in Pregnancy

BY

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It is recognized that all types of anaemia may complicate pregnancy, but aplastic anaemia is a very unusual associated disease. This case history records a temporary acute aplastic anaemia occurring late in pregnancy, with recovery after delivery.

Mrs. W. D., aged 36, 6-para (Case MM. 1814).

Previous obstetric history. Four normal pregnancies and confinements. The 5th (twins) was complicated by hyperemesis gravidarum and anaemia. During the 2nd month the blood count showed the red-blood cells to be 6,160,000 per c.mm. and the haemoglobin to be 83 per cent, but the red-blood cells fell to 3,190,000 per c.mm. and the haemoglobin to 60 per cent at the 5th month, and remained at this level. No further details are available except that she had a severe secondary postpartum haemorrhage.

Sixth pregnancy. The woman was first seen at the 14th week, when she seemed rather pale but had no symptoms. Haemoglobin 73 per cent, red cells 4,380,000 per c.mm., and colour index 0.83. She attended the borough clinic and was next seen at this hospital at the 35th week. She had had some swelling of the legs for 4 weeks, and diarrhoea and vomiting for 2 weeks, which had left her with extreme lassitude and breathlessness. She was at once admitted and found to have marked pallor and gross oedema of the feet. The blood-pressure was 120/70. The urine was normal. The uterus corresponded with the expected size, and the liver and spleen were not palpable. The blood-picture was haemoglobin 39 per cent, red cells 2,350,000 per c.mm., and colour index 0.82. Anisocytosis and hypochromic cells present. Rhesus factor positive. Blood group A. She was transfused with 540 c.cm. blood, which raised the haemoglobin to 44 per cent, but at the end of the first week her blood count had fallen to haemoglobin 33 per cent, red cells 1,840,000 per c.mm., colour index 0.91, white cells 1,000 per c.mm. (polymorphs 40 per cent, lymphocytes 56 per cent, monocytes 4 per cent), platelets 65,000 per c.mm. Sternal puncture showed a great reduction in the number of nucleated cells. The myelocytes were mostly degenerated, but there was an excess of eosinophils. Some giant platelets were seen. The myelogram excluded a megaloblastic anaemia of pregnancy and indicated an aplastic process. She was given 5,400 c.cm. blood within the next four days and her haemoglobin rose to 85 per cent. At this time she began to have bleeding from the gums, purpuric spots appeared on the arms, and within the next 3 days she had epistaxis, melaena and gross haematuria. The white cell count remained very low and fell to 400 per c.mm., but rose again to 2,000 per c.mm. before delivery. During this time she had daily injections of 4 c.cm. Hepalon intramuscularly, but reticulocyte response did not occur. She was also given iron, ascorbic acid and Aluzyme yeast by mouth. She was given 2 further transfusions (see chart), and then labour was induced by high rupture of membranes. She was delivered in 4 hours of a live child, weighing 6 pounds 10 ounces. She had a severe postpartum haemorrhage before and after expulsion of the placenta and was given 2,100 c.cm. blood. Within 3 days her white cell count had risen to 4,000 per c.mm., platelets to 190,000 per c.mm. Haematuria persisted for a few days. The bleeding time was over 15 minutes and the clotting time (Dale & Laidlaw) 1 minute 45 seconds. She had a *Bact. coli* infection of the urine, excessive lochia and pyrexia up to 100.6° F., but her general condition

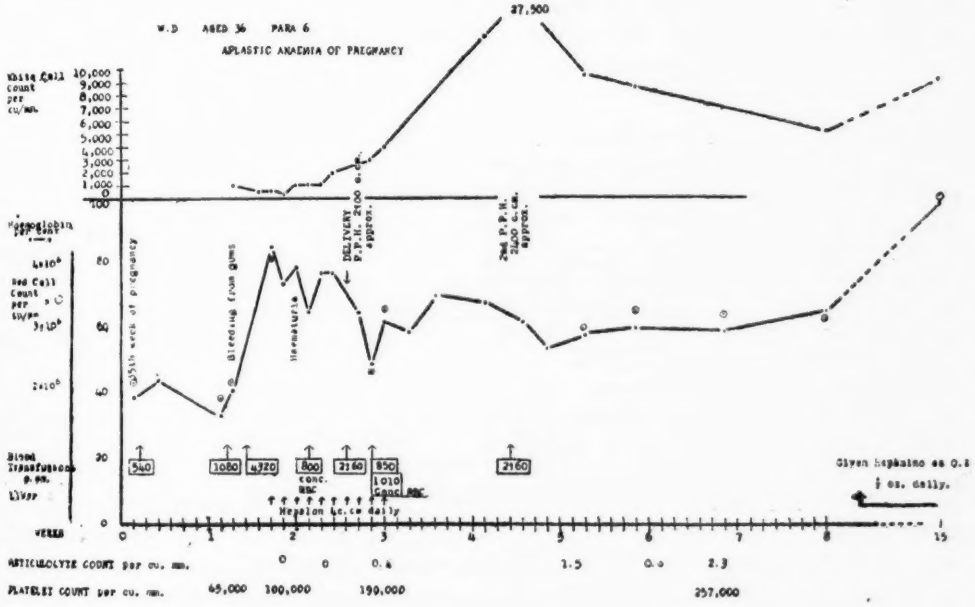
was greatly improved. Two weeks after delivery she had a sudden secondary postpartum haemorrhage and lost nearly 2½ litres of blood. The uterus was explored under anaesthesia and a placental polyp was removed. Blood transfusions of 2,160 c.cm. were given. After this she made a good recovery. By the 8th week the haemoglobin had risen to 80 per cent and the red-blood cells to 3,650,000 per c.mm., and she was sent home.

From the 7th week onwards she received proteolysed liver (Hepamino, ½oz. daily) and she continued to take Aluzyne Yeast, ascorbic acid and ferri et ammon. cit., and by the 13th week the haemoglobin was 98 per cent and the red cells 5,000,000 per c.mm. A fractional test meal showed a normal acid curve.

Sterilization was performed 6 months later, and 18 months after delivery she remains well with haemoglobin 80 per cent, red cells 4,800,000 per c.mm., white cells 6,200 per c.mm. (neutrophils 74 per cent, eosinophils 2 per cent, lymphocytes 21 per cent, monocytes 3 per cent, and platelets 484,000 per c.mm.

The baby was healthy. Blood count on the 4th day was haemoglobin 134 per cent, red cells 6,680,000 per c.mm., colour index 1.0. Rhesus positive. Blood group A. She was not breast fed owing to the mother's poor general condition after delivery.

The patient's husband was also Rhesus positive and blood group A.



Discussion.

This patient presented the features of an aplastic anaemia complicating pregnancy. The profound normochromic anaemia with reduction in all the cellular elements in the blood and the marked haemorrhagic tendency, together with the bone marrow picture, were consistent with this diagnosis. The absence of megaloblasts excluded the more common megaloblastic anaemia of pregnancy. The subsequent good progress of the patient is much against the possibility of a leucopenic myelosis.

A very careful interrogation was made concerning exposure to known potentially toxic substances such as benzol, arsphenamine, phenacetin, radio-active paint, X-rays, radium and others[1], but none could be

found. It is recognized that an idiopathic aplastic, or severe hypoplastic anaemia may complicate pregnancy[2,3,4], but only 14 cases have been reported, and of these the diagnosis is doubtful in 8 from the data available. They have been reviewed by Hurwitt and Field[5] and their table is appended with our case added. Only 1 was a true chronic aplastic anaemia; she had exacerbations during 3 pregnancies and improvement following termination on each occasion. The others were acute anaemias discovered during pregnancy or the puerperium. Of the 14 cases, only 5 recovered, and it is significant that in all these cases pregnancy was terminated before recovery took place. Our case confirms the beneficial effects of termination of the pregnancy.

	Age	Stage of pregnancy	Obstetric management	Outcome	Remarks
1. Ehrlich, 1888	21	First trimester	Abortion	Death	
2. Planchard, 1887	29	At term	Normal delivery	Death	Diagnosis questionable
3. Hanot and Legry, 1889	25	Second trimester	Miscarriage	Death	Diagnosis questionable
4. Ricca-Barbesis, 1908	25	Puerperium	Normal delivery	Death	Sepsis
5. Hirschfeld, 1906	34	First trimester	Abortion	Death	Gangrenous endometritis
6. Hirschfeld, 1906	38	At term	Normal delivery	Death	Died 9 months postpartum
7. Massary and Weil, 1908	21	First trimester	Uninterrupted	Death	Death due to heart failure
8. Jungmann, 1914	29	At term	Normal delivery	Recovery	Diagnosis questionable
9. Larrabec, 1925	30	First trimester	Uninterrupted	Death	No postmortem
10. Muller, 1938	28	Postpartum	Normal delivery	Recovery	Several transfusions
11. Foderl, 1938	23	Third trimester	Normal delivery	Recovery	Several transfusions
12. Nicuwenhuis, 1938	18	Third trimester	Interrupted	Recovery	Severe case
13. Dobriner, Rhoads and Mummel, 1938	30	Three pregnancies	Interrupted	Recovery	Chronic, with exacerbations
14. Hurwitt and Field, 1939	27	First trimester	Uninterrupted	Death	Severe case
15. Bigby and Jones, 1946	36	Third trimester	Interrupted	Recovery	Several transfusions

Summary.

A case of acute aplastic anaemia during the latter part of pregnancy is reported in a woman aged 36 years. Recovery occurred after massive blood transfusions and the induction of premature labour.

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A Home-made Repositor for Chronic Inversion of the Uterus

BY

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ON August 24th, 1944, a Bhil woman, aged 25, was brought to the Mission Hospital at Dohad. She gave a history of having been delivered of her first baby 5 days previously. The bowels had not been moved for 5 days, urine had not been passed for 4. She also described high fever with rigors on each of the previous 5 mornings. There had been slight abdominal pain for 4 days, no vomiting, and the lochia was scanty. The baby was alive and well.

On examination she looked very ill and was very anaemic. There was not any tenderness or rigidity in the abdomen but a midline mass, simulating the uterus, extended from the symphysis pubis to 2 finger-breadths above the umbilicus. The edge of the spleen was palpable on inspiration.

On admission to the ward the temperature was 101°F., pulse 120 and respiration 26. She was catheterized and relieved of 26 ounces of urine (acid; trace of albumen; no sugar) and put on a mixture containing quinine sulphate, gr. 5, t.i.d.

The next day the nurse drew my attention to a mass at the vulva which she discovered when doing the routine washing. The uterus was not palpable in the hypogastrium, and on vaginal examination I was surprised to find a large mass, which turned out to be the inverted uterus. The lochia was scanty, yellow and very offensive. An attempt to replace the uterus by manual taxis failed.

On further enquiry I learned that the patient had been 3 days in labour and that the baby had been born only after the men-midwives of the village had been called in and had exerted much pressure on the abdomen, according to their custom. As the placenta was slow in being born abdominal pressure had again been applied and the placenta was expressed and apparently the uterus with it. Thereupon the patient felt extremely nauseated.

Until August 28th the temperature varied between 100 and 102°F. but after this date it did not rise over 101° and swung as low as 99°. The pulse varied between 120 and 140.

On August 31st the quinine mixture was discontinued and sulphanilamide, 1 g., t.i.d., commenced.

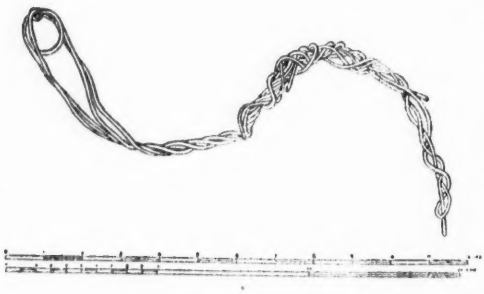
September 2nd. The 12-hourly catheterization was stopped as the amounts of urine which were being withdrawn for the previous 2 days were only 2 to 8 ounces, and the patient was passing urine voluntarily. The bowels were acting normally after a dose of magnesium sulphate on the morning following admission.

September 8th. The sulphanilamide was stopped because the temperature had not been above 99.4°F. since September 3rd, the pulse had steadied at 120 and the lochia had practically ceased. Blaud's pills, gr. 10, t.i.d., and liver injection, 2 c.cm. intramuscularly on alternate days, were commenced.

September 15th. I had written to Bombay, to a firm of surgical instrument makers, for an Aveling's repositor. But after 10 days I got a reply saying they had

none in stock so I decided to attempt to make something myself which, if possible, would avoid an alleged disadvantage of the Aveling model of which a textbook gave warning, viz., after replacement of the uterus has been accomplished there is said to be difficulty sometimes in getting the head of the Aveling repositor out of the uterus if the cervix contracts in behind it.

The model I evolved was simply an S-shaped contrivance made of galvanized iron wire, of 11 and 14 gauge, with a torpedo-shaped end to be applied to the uterus thus keeping the cervix sufficiently dilated to allow of the withdrawal of the repositor (see illustration).



After sterilizing this home-made repositor and after giving the patient a Dettol douche, I placed it in position against the inverted fundus and packed the vagina with a sterile gauze roll to keep it from slipping. Four bandages from the free end of the S tied to a bandage round the waist, which was steadied by another bandage brace-like over the shoulders, held it in position. With the bandages taut the springiness in the wire S exerted a steady pressure on the uterus. Elastic was not available.

September 16th. After 24 hours I removed the apparatus. The uterus was certainly being replaced but I was disturbed by the way the end of the torpedo, blunt though it was, had cut into the muscle of the uterine wall. Perhaps it was only to have been expected. As an improvement the torpedo frame was padded with sterile lint and cotton wool bound on with a sterile bandage, taking special care to put an extra amount of padding on the 'point.' Over this a rubber condom was drawn to keep the padding from becoming soiled and septic. This was tied at the bottom to prevent discharge seeping inside. Again over this a second condom was pulled and tied for additional security. Now it could not possibly cut into the uterine wall as the frame could not be detected through the padding. After a douche the repositor was reinserted and packed into position.

September 17th. On vaginal examination to-day the fundus was found to be level with the external os.

September 18th. For the past 4 days the temperature has been elevated but not exceeding 100°F., pulse 100. To-day the fundus is well inside the internal os. The repositor was removed and the torpedo end bent a little to lessen the angle it made with the shaft. After a vaginal douche it was replaced.

September 19th. To-day the inversion was found to have been completely replaced as determined by a bimanual examination with a finger inside the uterine cavity. Repositor discarded. To be douched daily.

September 20th. Temperature 98.4°F.; pulse 100 yesterday and to-day.

September 26th. On bimanual examination the uterus was found to be well involuted. Cervix closed.

September 28th. Discharged. Temperature and pulse normal for the past 7 days.

I am indebted to Mr. Leslie Baxter for the drawing of the repositor, made after it had achieved its purpose.

INDEX TO CURRENT LITERATURE

In this Index an endeavour will be made, so far as conditions permit, to maintain an up-to-date record of all titles bearing on obstetrics and gynaecology and the infant. The classification aims at making immediately available for the reader the titles under the various subject headings. In the majority of instances the title is obtained from the original publication. Where this has been impossible, the title has been obtained from the abstracting journal shown in brackets. Reprints will be welcomed and should be sent to the Editor.

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